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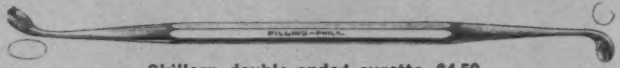

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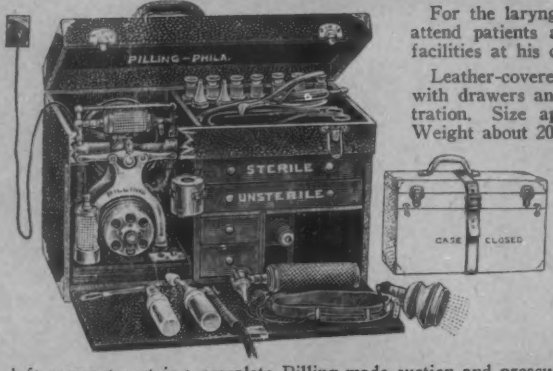


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THE

# LARYNGOSCOPE.

VOL. XL, MARCH, 1930. No. 3

## ORIGINAL COMMUNICATIONS.

(Original Communications are received with the understanding that they are contributed exclusively to THE LARYNGOSCOPE.)

### SOME EXPERIENCES IN THE SURGICAL TREATMENT OF INFLAMMATION OF THE FRONTAL SINUS, AND ITS COMPLICATIONS.*

DR. HERBERT TILLEY, London.

*Mr. President and Gentlemen:* My first and grateful duty is to thank the Council of this distinguished Academy of Ophthalmology and Otolaryngology for asking me to be your guest of honor on this occasion.

You, Sir, were kind enough to suggest that I might relate some of my experiences in the surgery of the frontal sinuses and of the more serious complications which may attend operative intervention for the relief of inflammatory conditions.

I accepted your choice because all pioneers are delighted to look back, and the inexorable finger of Time points to an article in *The Lancet,* Sept. 26, 1896, entitled "An Investigation of the Frontal Sinuses in 120 Skulls with Cases Illustrating the Methods of Treatment of Disease in This Situation." Again, in the *British Medical Journal,* 1899, there may be found what I believe to be the first description of osteomyelitis of the frontal bone following upon an operation for chronic suppuration in its sinuses. Unfortunately, the subject of that communication died but I am able to show you the calvarium removed at the postmortem examination.

It would appear to be an almost unique specimen, but I trust that none of you will ever experience the misfortune of being able to secure a duplicate.

*Guest of Honor's paper read before Amer. Acad. of Ophth. and Oto-Laryn., Atlantic City, Oct. 22, 1929.

Editor's Note: This ms. received in Laryngoscope Office and accepted for publication March 3, 1930.

These early communications were published rather more than 33 years ago and by a lone young "tenderfoot" in otorhinolaryngology whose presence before you today he regards as a signal compliment to one of the early British workers in the revival of the surgery of the nasal accessory sinuses.

Before entering into any details of our subject it may be well to state some general principles or considerations which the surgeon must keep in mind when he undertakes the responsibility of treating acute or chronic inflammation of the frontal sinus by any form of surgical intervention.

*1.* He must have an accurate knowledge of the anatomy of the sinus, of its commoner variations and of its relation to important surrounding structures.

*2.* He must never forget that inflammation is a protective reaction on the part of the tissues in their effort to resist and overcome a noxious invader, and therefore every detail of our treatment should aim at a minimum of trauma commensurate with such assistance as we may be able to afford Nature in her struggle for restitution.

*3.* If this conception be granted, then it will be obvious that our primary object in surgical treatment will and must be the establishment of free, spontaneous and permanent drainage of the products engendered by both invader and defender. I have said "free, spontaneous, permanent drainage"—four words—and would urge the younger members of our specialty to write them on the tablets of their hearts, and bind them on their foreheads.

In the treatment of inflammation of the frontal sinus our paramount difficulty is that we are dealing with an infected mucous membrane which lines an irregular and noncollapsible, bony-walled cavity from which drainage is impeded or, on occasion, completely obstructed.

To make this free and spontaneous has been the object of every operation yet devised, whether it be by the external or by the intranasal route. Had any one of them been uniformly applicable or successful, we should not be discussing the subject today. So we ask ourselves, "Wherein lies the difficulty with an external operation which is not so pre-eminent in any other inflamed bony cavity of the body?"

It seems to me that the answer is obvious, *viz.*, the patient has a natural and instinctive dislike of a striking and disfiguring deformity.

Hence it comes about that our ideal operation must not only provide for free drainage—the basis of cure—but this must be attained with the best cosmetic result.

How can we secure these ends in acute and chronic inflammation of the frontal sinus?

ACUTE INFLAMMATION.

If we are fortunate enough to see the patient before the infection has destroyed the mucoperiosteal barrier and entered the intimate bone structure of the sinus wall, it is often possible to encourage free drainage into the nose by the application of cocain, adrenalin or ephedrin solutions to the lower end of the frontonasal duct where it opens into the anterior region of the middle meatus. Such a measure, possibly supplemented by scarification of the adjacent mucous membrane, will often suffice to give the desired exit for inflammatory secretions.

Should this fail, removal of the anterior half of the middle turbinal followed by frequent irrigation of the sinus with a warm hypertonic saline solution will often effect the freer drainage which is aimed at. If it be found that the nasofrontal canal will barely admit an ordinary probe, it may be well to slightly enlarge the canal and ostium in an anterior direction with a suitable rasp or burr. The value of irrigation is that it removes the irritating products of bacterial activity and of those incident to the inflammatory reaction of the mucosa. Such evacuation will often relieve the chief subjective symptom—headache—and what is also important, it will tend to reduce the edematous obstruction around the frontonasal duct. When inflammation has passed into and through the bone to reach the periosteum it discloses itself by edema of the soft tissues over the anterior wall of the sinus or of the upper eyelid, and in these circumstances it is unlikely that anything but external operation will suffice. This should not be unduly delayed because we can never say when a subperiosteal abscess may not bring about a septic thrombosis of an ophthalmic vein (superior or inferior) leading toward the cavernous sinus, with possible infection of the meninges.

EXTERNAL OPERATION.

The type of incision for exposing the junction of the anterior wall and the upper and inner circle of the orbit is known to you all. When a disc of bone has been removed by a small trephine or by hammer and gouge, the aperture may be carefully enlarged by Citelli's or other suitable forceps, the limits of the sinus ascertained by a probe and its inflammatory secretions gently washed out by a cannula. If necessary, the ostium and frontonasal canal can be enlarged by a suitable probe or burr passed from the above downwards. Three to four small drainage tubes should be inserted

so as to project through the middle of the external wound, and finally removed, when a mucoid discharge proves that the inflammatory process is practically at an end. For the first day or two hot fomentations should be frequently applied over the affected sinus because they relieve pain and assist in resolution of inflammation in the soft tissues. Under the influence of warm daily irrigations and later by injections of a 15 per cent argyrol solution the mucosa will gradually return to normal. The tubes can then be dispensed with and the external wound may be allowed to close.

It has been my good fortune not to have had a case of postoperative meningitis or of osteomyelitis follow the external operation for acute inflammation of the sinus, but I have published an instance of spontaneous osteomyelitis in an untreated case (*Brit. Med. Jour.*, July 7, 1917).

Certain difficulties may confront the surgeon in his endeavor to establish free internal (intranasal) and external drainage. I will mention two only:

*1.* A tortuous and narrow frontonasal canal. This one would attempt to overcome by a suitable burr passed from above downwards, or by removal of a portion of the ascending process of the maxillary bone and of the adjacent lacrimal bone.

*2.* A very large, irregular, and possibly loculated sinus. In such cases I should adopt a procedure which will be described later for the radical operation in chronic suppuration.

### WARNINGS.

*a.* I can conceive of no circumstances in which a sharp curette should be used in removing acute or chronic inflamed mucous membrane from an infected frontal sinus. It is so easy to uncap the adjacent diploe, which are probably already inflamed, and by the admission of septic organisms risk a spreading and nearly always fatal osteomyelitis. If it be really necessary to remove masses of edematous mucosa, this should be done with a mop of sterilized gauze or with blunt forceps, such as those introduced by Luc, Paris.

*b.* Whenever a general anesthetic is administered for operation on the frontal sinus, the postnasal space should be packed with a sterilized sponge in order to prevent blood and septic secretions passing into the lower air passages. Of course intratracheal ether anesthesia will obviate the need of such a precaution.

### CHRONIC FRONTAL SINUS SUPPURATION.

Before discussing the operative treatment of this condition, I would like to make a few statements which embody some personal experiences.

*1.* Headache is the most important and significant of the subjective symptoms. Its response to treatment will favor a good prognosis, whereas its persistence after intranasal operation may raise the question of external intervention.

*2.* The removal of the ethmoidal infection which is practically always present, at any rate in the anterior cells, is the key to success in any type of internal or external operation. It is almost of equal importance to drain infected antral and sphenoidal sinuses before, or at the same time as the frontal sinus operation.

*3.* A practical point—it is often easier to pass a probe or cannula into a chronic suppurating frontal sinus than into one under normal conditions.

*4.* No operation should be undertaken without having studied frontal and lateral radiograms of all the sinuses. Their evidence may be the sole deciding factor in determining the particular type of operation to be performed.

## TREATMENT.

If an experience of more than a quarter of a century has taught me anything of this subject, it is that the number of cases of chronic suppuration of the frontal sinus which call for external radical operation are few.

In making this statement, let me say that I am not forgetful that a focus of sepsis in that situation has close anatomical relationships with the brain and its meninges and with the contents of the orbit. The purulent discharge may find its way into the larynx, trachea and lower air passages with the evil results we are familiar with—or it may be swallowed into the stomach with a sequence of gastro-intestinal disorders.

Carried by the bloodstream or lymphatics, the micro-organisms or their toxins may produce lesions in the large or small joints, in the cerebrospinal nervous system, or failing such lodgment, symptoms of a general toxemia may be revealed. A dark enough picture indeed, if, in its contemplation we forget the existence in the body of Nature's immunizing agents and her wonderful powers of defense.

In my earlier practice I saw many patients with chronic frontal sinus suppuration and often advised some external radical operation. Frequently such treatment was refused in favor of simpler palliative measures. Today, many of those people are old men and women, who have enjoyed healthy, happy and useful lives. They visit me occasionally for inspection, for the solace of an optimistic report, or for some simple intranasal treatment.

It has interested me to note that in some of them the discharge has entirely ceased, in others it is so slight as to form a thin, non-fetid crust in the anterior ethmoidal region, which the patient can easily expel by a simple alkaline nasal wash. Had I performed a radical external operation on these cases, they would all have had some degree of scarring, probably the discharge would not have been cured in many of them, and in a few some serious complication might have supervened.

Such an experience seems to be in accord with that well known line of Tennyson: "Knowledge comes but Wisdom lingers," which is another way of stating that Youth fixes its gaze on possibilities, while maturer years constrain us to regard the broader field of probabilities.

We may now pass to a brief outline of the chief operations which have been introduced for the relief or cure of chronic suppuration.

For the reasons already given, it seems to me that unless these are definite indications to the contrary, the intranasal operation should be given precedence in our attempt to cure or to relieve the patients' symptoms.

In the average type of case these are headache, pain on pressure over the anterior or anteroinferior vault of the sinus, and not infrequently some degree of nasal obstruction caused by polypi or other high-grade mucous membrane hypertrophies. The diagnosis is established by intranasal examination and supplemented by radiography.

The intranasal operation about to be outlined was introduced by Schäffer, developed by your countryman, Fletcher Ingals, and again by Halle, of Berlin, and many others.

### THE INTRANASAL OPERATION.

This involves removal of the anterior half of the middle turbinal at its junction with the "lateral mass," of any polypi or mucous membrane hypertrophies in the middle meatus and around the frontonasal canal, and possibly enlargement of the latter and of the ostium of the frontal sinus.

This seems a fitting opportunity for saying how much rhinologists must be indebted to you, Mr. President, for pointing out the surgical importance of the "agger" cells and for demonstrating how their removal may greatly facilitate the approach to the infundibulum and the establishment of freer drainage from the sinus.

For the enlargement of the frontonasal canal I employ a rasp with serrations on its anterior surface only, so that the canal and ostium are enlarged solely in a forward direction. Our chief difficulty lies in reducing the prominence of the frontal "boss" which

forms the main hindrance to free drainage from the sinus. I have seen Halle, of Berlin, use his electrically-driven burr for this purpose, and was struck by the width of the communication which is secured. Having no personal experience of the method, it would be unfair to offer any adverse criticism of it; but fatalities have been recorded after its use, and in any case I have an instinctive preference for an instrument manipulated by the hand and guided by that indefinable but intimate sense of touch. The last step of the operation will be the removal of any tags of mucous membrane or semidetached spicules of bone. As free drainage is the great desideratum, no tampons of gauze should be inserted in the proximity of the operated region.

Three days later it is my practice to wash out the sinus with warm boracic lotion or a mixture of normal saline and peroxid of hydogen. When the discharge lessens and becomes more mucoid, a 15 per cent solution of argyrol is injected after a preliminary irrigation with one of the above lotions.

For a few days after the operation the discharge may be profuse because the drainage has been improved, but if the headache has been relieved there should be no hurry to contemplate any external operation because it is reasonable to hope that the other symptoms will gradually abate, even though this may involve some weeks or months of waiting.

To the rhinologist who is skilled in intranasal surgery the operation is a comparatively easy one, and while serious and even fatal complications are by no means unknown, they are much less frequent than with the more radical external operations. This fact, together with the absence of any scar or deformity, the short demands which the intranasal procedure makes upon the time of the patient, the great relief and occasional cure of the symptoms which may be expected from it—all these embolden me to make the statement that in seeking to "make the punishment fit the crime," with a few exceptions the intranasal route should always be chosen in the first instance.

In this profession of my faith I am glad to find that Prof. Hajek, of Vienna, in the last edition of his classic work says: "I am convinced that in the majority of uncomplicated cases of chronic frontal sinus empyema, resection of the middle turbinal and the removal of hypertrophies is usually sufficient."

We may now ask ourselves, "What may be the conditions or the complications which would constrain us to advise a primary or a secondary external operation?"

PRIMARY EXTERNAL OPERATION.

*1.* A very narrow nasal cavity preventing free access to the ethmoidal region.

*2.* A tortuous frontonasal canal which proves to be impassable by a suitably curved probe, even after removal of the agger or other anterior ethmoidal cells in the neighborhood of the canal.

*3.* An extensive, irregular and loculated sinus. Not infrequently such secondary intrafrontal sinuses are associated with frontoethmoidal cells which spread outwards between the roof of the orbit and the floor of the frontal sinus. It will be obvious that the most perfectly executed endonasal operation could not provide effectual drainage for such conditions.

*4.* Attacks of subacute periostitis of the anterior or of the anteroinferior wall of the frontal sinus associated with tenderness on pressure over those regions and possibly with edema of the upper eyelids. In this category one may place a chronic external fistula opening below the supraorbital ridge. Such conditions imply subperiosteal suppuration of the frontal or frontoethmoidal sinuses, and we can only deal with them effectually by an external operation which will enable us to remove carious bone and establish free intranasal drainage. On the other hand, I can recall a few cases of recurrent periostitis, one of chronic external fistula in an old man, and two cases of recurrent iritis which were cured by endonasal operation, but I regard them as instances of good fortune and would only employ such a method in exceptional circumstances.

Time will not permit me to discuss the big and vexed question of the relationship between retrobulbar neuritis and frontal sinus or frontoethmoidal suppuration. I will only say that the nerve lesion is more likely to be associated with ethmoiditis than with the frontal sinus inflammation, and possibly the best results *qua* the optic nerve have resulted from operations on an apparently normal ethmoid or sphenoid sinus. Nevertheless, it would seem wise that, in the presence of a serious and progressive intraocular or retrobulbar lesion, an obvious focus of sepsis in any paranasal sinus should not be left untreated.

*5.* Symptoms of subdural abscess will demand a radical external operation and a suspicion of this complication may be strengthened by radiography of the frontal sinus regions.

SECONDARY EXTERNAL OPERATION.

*1.* When intranasal measures have failed to relieve the chief symptoms, *e. g.,* headache and profuse discharge, especially if this be fetid.

2. The desire of the patient to be freed from the discharge which has not been cured or minimized by endonasal operation.

Other less common conditions may be symptoms suggestive of subdural abscess, or those which have been termed by William Hunter the "septic psychoses." You will probably add to this list the distal manifestations of sepsis as they affect the joints, the peripheral nervous system, the lower air passages, and the gastrointestinal tract, or the effect of blood-borne toxins on the general health.

EXTERNAL RADICAL OPERATIONS.

*What type of operation should be selected?*

Let me say at once that there is no single operation which will meet the requirements of every case. It might be so if we could obliterate the sinus entirely as advised by Riedel, but cosmetic considerations will often preclude such a procedure. On the other hand, if radiography proves it to be of average size and shallow from before backwards, its obliteration will produce a cure with almost unnoticeable scar of depression. The operation entails removal of the anterior wall and the inflamed mucosa, establishing a free passage into the nose and encouraging the cavity to fill with healthy granulation tissue before the external incision is allowed to close.

Many of my most satisfactory results have been secured, in selected cases, by this method of obliterating a sinus. About six weeks of daily after-treatment will be required for a sinus of average dimensions before complete healing has taken place by granulation tissue. This seems to me to be the point most open to adverse criticism. It resembles Coakley's operation, and differs from Kunt's procedure in that while I have only made one incision below the eyebrow and curving downwards towards the internal canthus, he cut a triangular flap in order to expose the anterior wall and inserted a drainage tube at the point where the vertical and horizontal incisions joined one another, and with the exception of this opening he sutured his incisions at the close of the operation.

I have had no experience with the Ogston-Luc operation, *viz.*, opening the sinus at the junction of its lower anterior wall with the upper and medial wall of the orbit, curetting the mucous membrane and the anterior ethmoidal cells, placing a drain from the sinus into the nose, and suturing the incision at the close of the operation. The restricted opening does not give a sufficiently wide field for inspection of the sinus cavity, the establishment of free intranasal drainage must often be an almost blind manipulation and immediate closure of the external wound probably accounts for many of the fatal complications recorded in the literature of the subject.

Jansen preserved the anterior wall of the sinus but removed its floor before curetting away the mucous membrane. It is an excellent method for a comparatively small sinus and affords access to the ethmoidal cells, but in a large sinus with many recesses it is often difficult to reach these. Jansen acknowledges this himself and admits that it may be necessary to encroach on the anterior wall.

Riedel recommended the complete removal of the anterior wall and the floor of the sinus, which is undoubtedly the best method for complete obliteration of the cavity and for exenteration of the ethmoid—but, as I have said already, the resulting deformity would frequently render such an operation impracticable.

Killian endeavored to overcome these difficulties by his well known "bridge" operation. Many of us practiced it, but in a sinus of considerable anteroposterior depth its obliteration by granulation tissue failed to take place, a dead space resulted and suppuration frequently recurred. Furthermore, not rarely the bridge necrosed and gave way, with a resulting deformity which it was one of the chief objects of the operation to prevent. In favorable cases, however, the operation gave excellent results as regards the symptoms, as well as a minimum of deformity.

Like most other operations fatalities occurred from meningitis or osteomyelitis. Hajek collected 23 recorded cases. Who knows how many more have remained unpublished?

In a large sinus with many recesses and extensive ethmoidal disease, the operation which is most frequently practiced in London is that devised by Walter Howarth (*Jour. Laryngol.*, Vol. 36, No. 9, 1921, p. 417). It provides for very free intranasal drainage by removal of the frontal "boss" and part of the ascending process of the superior maxillary, easy access to the ethmoid and sphenoid sinuses and the cosmetic results are excellent.

To save time, I will show you the essential details of the operation by means of slides which have been taken from his own illustrations.

You will note that no attempt is made to remove all the inflamed mucosa, because for its ultimate restitution dependence is placed on free, spontaneous and permanent drainage.

My personal experience of the method is sufficiently satisfactory to prompt my recommending you to give it a fair trial, if you have not already done so. Having had no personal experience of Ritter's, Knapp's and Lothrop's operations, nor of osteoplastic resections, it would seem a waste of time and scarcely fair to criticize them.

THE COMPLICATIONS OF OPERATING ON THE FRONTAL SINUS.

It will be necessary to pass over all but a few of the less dangerous results in order to give more time to the serious risks of surgical intervention.

*Diplopia* is frequent after any operation which interferes with the pulley of the superior oblique muscle, but the trouble is generally temporary and in all my experience, in only one case has the symptom remained permanent.

*Postoperative Orbital Cellulitis:* This may complicate internal and external amenable to free incision and provision for drainage followed by hot fomentations until edema tends to subside. In no instance have I known it to cause permanent damage to the sight.

*Anesthesia* will of course always follow division of the supra-orbital nerve. Formication in the area of distribution of the nerve will last many months and some degree of anesthesia will be permanent. I have never known division of the nerve to produce more than a temporary neuralgia.

The two most serious complications, because they are nearly always fatal, are septic meningitis, and diffuse, spreading osteomyelitis of the frontal bone.

As a general rule meningitis follows from infection starting in the ethmoid bone, but as inflammation of that structure is to be the subject of a special discussion, we may leave its complications until then. It need only be said that acute meningitis may occur as a direct result of radical operations on the frontoethmoidal sinuses or it may prove to be the fatal element in cases of osteomyelitis of the frontal bone. My friend, Logan Turner, has lent me some slides which show how septic meningitis may arise by infective organisms entering the lymphatic sheaths of the olfactory nerve expansions.

DIFFUSE OSTEOMYELITIS.

This, the most serious complication of operations on the frontal sinus, has been so fully and ably described by my friend, Dan McKenzie, in the *Journal of Laryngology, Rhinology and Otology* for January, February and March, 1913, that it would be impossible for me to improve on it even if time permitted my attempt to do so. It will suffice if I make a few general observations on the complication before discussing its prophylaxis and treatments.

Its chief characteristic is the tendency to spread beyond, and often far beyond, its source in the effected sinus. The calvarium I have shown you exemplifies this fact.

Staphylococcus (albus and aureus) or streptococci are the organisms most frequently found in pus from the inflammatory foci in the

bone; the pneumococcus rarely, and in one case McKenzie established the presence of B. coli. He remarks that "so far as the morphological type of the organism is concerned, no light whatever is thrown on the etiology of the disease." Whether such organisms gain access primarily into the osseous tissue around the sinus, or whether efferent veins are first infected and the bones around them secondarily implicated, are questions as yet unsettled.

From observation of the clinical features of osteomyelitis it is my impression that both portals share in providing access for infection.

But the riddle which we want to solve is this: How does it come about that experienced, skillful and careful surgeons may, after a long series of successful results, be suddenly brought face to face with this night mare complication which may be followed by another run of happy results? For example, Killian performed his operation on 86 cases without accident, but lost three, one after the other, before his hundred were completed! It is difficult to believe that in those three fatal cases the infective organisms were peculiarly virulent, or that the resistance of the patients was out of all proportion so much lower than in the more fortunate cases.

Before passing to prophylaxis, I would like to state that I have lost one patient from a spreading osteomyelitis which started (after a tooth extraction) in the right antrum and spread upwards to the frontal sinus. After many extensive operations in the attempt to check its progress, the patient succumbed to a basal meningitis.

Another case followed a bilateral intranasal operation for chronic suppuration of the frontal sinuses. When these were opened up a chronic subdural abscess was found behind the posterior wall of the left sinus. This may have been the cause of what the patient called his "devastating headache"—the symptom for which the preliminary intranasal operation was performed.

### PROPHYLAXIS.

If we are to avoid this complication, I am convinced that:

*1.* Before operation on the frontal sinus we should see that there is no untreated sepsis in any of the other sinuses, and especally if the antrum and the ethmoidal cells.

*2.* Immediately before the anesthetic is administered, the sinus should be thoroughly irrigated so as to minimize the risk of contaminating the exposed edges of the bony wound.

*3.* When the sinus cavity has been exposed, any remaining purulent discharge should again be washed out.

*4.* The less the mucous membrane is interfered with the better. High-grade hypertrophies may be removed with blunt forceps, such

as Luc's, or possibly detached by small swabs of sterilized gauze. In no circumstances should a sharp curette be employed, especially in the upper regions of the sinus, where already inflamed diploe may easily be opened and infection admitted.

5. At the close of the operation only the extremities of the skin wound should be sutured; the central portion should be left open for three or four days, and the loose stitches then tied.

Drainage can be secured by a parallel series of four or five small rubber tubes leading from the sinus to the middle of the skin incision.

TREATMENT.

When once recurrent suppuration takes place in the wound and an area of pale, putty-like edema appears in the soft tissues overlying the neighborhood of (generally) the upper limits of the sinus, there must be no delay in making a free exposure of the frontal bone from the eminence to the affected sinus.

Free removal of the whole thickness must then be made with freshly sterilized instruments and commencing from above downwards, *i. e.*, from healthy towards inflamed and infected bone. Only by such drastic measures have a few threatened lives been saved.

Let me warn the inexperienced that the earlier phases of osteomyelitis come almost as unheralded as a thief in the night. There may be little or no headache, possibly no pyrexia, or only a slight rise to 99°, and the patient may not experience any general malaise or marked local discomfort—all these come later and the surgeon must not feel secure in their earlier absence. The pale and painless spreading edema around the wound must be to him the red flag of warning.

If and when such removal of bone has been made one or more isolated patches of edema should appear beyond the field of operation, it will be obvious that the surgeon's effort has been forestalled by septic thrombosis of a diploic vein and further intervention—though possibly worthy of consideration—is unlikely to be successful.

In these circumstances and dismayed with the sense of failure, he may be encouraged by the sentiments expressed by Tennyson in the last lines of his poem, "Ulysses":

> "That which we are, we are,
> One equal temper of heroic hearts
> Made weak by time and fate, but strong in will
> To strive, to seek, to find, and not to yield."

University College Hospital.

# ANALYSIS OF 100 CONSECUTIVE NASAL SINUS CASES TREATED CONSERVATIVELY.*

DR. EUGENE R. LEWIS, Los Angeles.

For the purpose of "taking stock" concerning results of distinctly conservative treatments applied to the average "mine run" cases of nasal sinus trouble, I made a detailed analysis of 100 consecutive private cases seen since January, 1926. Many of the cases in this series had been seen at other times for some reason or other; some of them had been under occasional treatment because of sinus trouble.

In attempting to evaluate the evidence furnished by these cases, many difficulties were encountered. To assemble with any degree of accuracy data respecting effects of treatment is quite different from assembling surgical data; for example, concerning operative recoveries, the time required for healing, or mortality percentages. Impressions as to improvement have been based upon the findings upon physical examination, gain in strength and weight, together with the patient's reports as to relief of symptoms. In addition to upper respiratory examinations, the majority of records include blood pressure, urinalysis and general physical observations; also allergic, metabolic, cardiographic and other findings in certain cases.

The cases of this series presented, as cardinal symptoms of sinus diseases: *1.* Recurrent attacks of nasal inflammatory symptoms; *2.* local pain; *3.* mucoid or purulent nasal discharge; *4.* hypertrophic, atrophic, fibrotic, polypoid or necrotic lesions of the mucosa; *5.* turbinal turgescence; *6.* gross architectural alterations of nasal structures; *7.* headache, frequently of morning type; *8.* aprosexia; *9.* disturbance of lower respiratory, gastrointestinal, glandular or nervous systems; *10.* general debility.

There are records of blood findings in 38 per cent of these cases; among other findings were syphilis, brucella abortus and plasmodium malariae; primary anemia was discovered in one case, and agranulocytosis in one case. These findings, together with the differential leukocyte counts, are not included in the tabulation because they are of no statistical value, in view of lack of any record of blood examination in 62 cases.

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The majority, 80 per cent, were seen in acute exacerbations of pre-existent sinus trouble. Sixty-three cases had been previously operated, from several months to several years before. Attempts to chart operative effects on the patient were finally abandoned, after considerable effort, as it became apparent that any such statistical evidence would be so questionable as to be of no value.

Of this series, 65 were in full possession of tonsils; 32 of these gave no evidence, in history or findings, of tonsillitis; 14 more had some remnants of tonsils, or regrown adenoid tissue in the tonsil regions, without apparent trouble therefrom.

Systolic readings below 100 are recorded as hypotension; systolic readings above 150 are recorded as hypertension; no records of blood pressures were found in the records of 16 cases below the age of 18 years; the tension percentages being figured on 64 cases over 18 years of age.

In 39 cases there was evidence of ("allergic") special tissue-sensitivity to foreign protein. This evidence included history of respiratory, skin, or both, reactions to certain foods, of family sensitivity and of individual sensitivity; in many cases allergic studies had been made, but because of wide variations in these test-methods, attempts to incorporate the details into the charts proved impractical.

In 18 cases, no pus was found in the nose on examinations; cytologic examinations by Sewall's technique were not made, the diagnosis of irritative or inflammatory disease of the lateral nasal wall tissues being established by gross findings and history; most of these showed occasional watery or glairy discharge, frequently also irritation of the transitional and skin areas at the introitus, in addition to cardinal symptoms of sinus disease. No etiologic relation is inferred between sinus disease and thyroid changes noted, though definite relations seem to exist between thyroid changes and hypertension. Omitting the general systemic febrile symptoms associated with acute phases of infectious sinus inflammation, there was little significant indication of commensurate etiologic relation between general systemic debility and sinus disease; that is, many cases showed local evidence of extensive suppuration and disease with relatively less systemic debility, and vice versa.

Thirty-two cases showed associated gastrointestinal disturbance, some of which were probably gastrointestinal expressions of the same tissue-sensitivity which, in the upper respiratory areas, was manifested as sinus trouble.

Almost 20 per cent showed chronic middle ear pathology; 13 per cent were arthritic; 4 per cent had associated pyelitis, only one of whom was an adult.

Morning headache was complained of in 52 cases; this varied from slight pressure and general "wooden-headed feeling" for an hour or two, to increasing headache up to 10 or 11 o'clock, subsiding by from 1 p. m. to 5 or 6 p. m.; 90 complained of difficulty in concentrating attentions for varying periods after rising.

After many failures to find some satisfactory expression of findings and results in the form of charts, attempts to express them statistically had to be abandoned, because in each chart there appeared misleading numbers and percentages. The following "digest of findings" contains in tabulated form the outstanding items encountered in this series.

DIGEST OF FINDINGS.

Ages 7 to 72; average, 37 years. Monolateral, 4; bilateral, 46; acute, 1; acute exacerbation, 80; chronic, 14.
TONSILLITIS—37 cases; acute, 10; chronic, 27.
PHARYNGITIS—Acute, 3; chronic lateral, 78; follicular, 86.
GINGIVITIS—Acute, 3; chronic interstitial, 38.
TENSION—Hyper, 10; hypo, 22.
THYROID—Palpable, 22; hypertrophy, 18.
EAR—Canalitis, 12; tympanitis acute, 3; chronic, 19.
EYE—Ant. uvea, 9; conjunct., 30; nerve, 1.
ARTHRITIS—Acute exacerbation, 6; chronic, 13.
HEADACHE—Acute morning, 52; chronic occasional, 20.
GASTRO-INTEST.—32.
PYELITIS—4.
SENSITIVITY—Individual, 19; family, 20.
PURULENCE—71; acute purulent, 11.
POLYPOSIS—Pellucid, 4; fibrotic, 3; mixed, 5.
HYPERTROPHY—Simple, 16; polypoid, 5.
ATROPHY—Slight, 11; moderate, 5; marked, 7.
PREVIOUS OPERATION—T-A, 35; septal, 14; sinus, 72; multiple, 58.
LOWER RESPIRATORY—Trach.-bronch., 25; bronchial, 9; asthma, 11.
CRANIAL NERVE—Retrobulb., 1; VII, 1; VIII, 2.
MENING.-THROMB.-ABSCESS—None.
OSTEOMYELITIS—None.
APROSEXIA—90.

RESULTS.

| | 30 days | 60 days |
|---|---|---|
| Disappearance of gross mucosal inflammation | 68% | 18% |
| Disappearance of sinus pain | 78% | 10% |
| Disappearance of mucopurulence | 64% | 6% |
| Disappearance of turbinal turgescence | 80% | 6% |
| Disappearance of crusting or ulceration | 68% | 6% |
| Disappearance of headache | 38% | 28% |
| Disappearance of aprosexia | 58% | 31% |
| Gain in weight, 52 cases | 28 | 10 |
| Improved blood pressure, 39 cases | 12 | 13 |
| Increased physical strength | 79 | |
| General improvement | 79 | |
| Intracranial complications | None | |
| Osteomyelitis | None | |

The table entitled "Results" sets forth certain items of improvement; no definite connection could be established between "findings," either individually or in groups, and "results." Many attempts were

made to devise certain correlations of results with findings, but each had to be discarded as it became apparent that nothing definite materialized. The relations between general systemic manifestations of pathologic processes and their local nasal manifestations are of particular importance; the relations between sinus inflammations and sensitivities (allergies) are of equal importance; concerning these relations statistical analysis of the data furnished by this series of cases was too vague to be charted. The inclination to attribute certain results to certain treatments leads to the fallacies notoriously associated with "post hoc-propter hoc" deductions. Above all, this attempt to analyze records emphasizes the lack of any measuring stick which may be used gauging results following treatment.

The tabulation of "results" is made up of information assembled from the individuals constituting this series of cases, and should be clearly understood as attempting to establish nothing further than a chronicle of changes observed. The improvements noted, regardless of the specific reasons therefor, have been highly satisfactory.

In 11 per cent the upper respiratory tract was apparently the site of *local pathology only*, whereas in 62 per cent there were acute exacerbations of chronic upper respiratory dysfunction, which were clearly and significantly *local* manifestations of *generalized pathology*, evidenced by changes in white cell count, temperature and metabolism; also in associated tissue reactions in other localities — lower respiratory, circulatory and gastrointestinal tracts, ocular and aural tissues, tendons, synovia, lymphatic tissues and certain gland structures.

It seems that many of the disturbances which are commonly termed "infectious inflammation of the nasal sinuses" are not limited to nasal sinus tissues, even though the patient's chief complaints are directed to the nasal areas. Etiologic factors of different natures are involved, and these different factors manifest their effects in different places and phases during the pathologic disturbance. The incidence of *acute nasal* symptoms is almost always brought on by shock, exposure, chill, trauma, fatigue, digestive disturbance or contact with noxious environment. Evidence of *systemic disturbances* succeed the onset of local nasal symptoms after relatively short intervals.

The nasal disturbance is of the nature of a reaction between nasal tissues and micro-organisms, while the succeeding, systemic disturbances are reactions between more remote body tissues and noxious emanations from the nasal areas. The nasal tissues in 90 per cent of this series were unquestionably in contact with pathogenic micro-organisms during the relatively symptomless periods between acute

exacerbations. There is apparent a somewhat uniform sequence of pathogenetic episodes in a large number of cases. Acute sinus trouble is practically always precipitated by some *physical* disturbance of normal working conditions of the body. The local changes of a *bio-chemical* nature which follow are signalized by disturbances of hitherto quiescent relations between nasal tissues and their micro-organismal residents. This is explainable only upon a basis of alterations *in the nasal tissues themselves* as it is inconceivable that chilling the ankles or breaking the femur will deposit new bacterial flora in the upper or lower respiratory areas to account for acute sinusitis or pneumonia. Wright[1] has said that "the skin is no less the cause of dermatitis than the tubercle bacillus is the cause of tuberculosis; without tubercle bacillus one does not have tuberculosis; without one's skin—well, there is no use wasting breath on that." There seems to be good reason for believing with Piness[2] that some infections take place when opportunities are afforded by acute allergic nasal reactions, regardless of bacterial nature of the allergy. In these observations is contained much food for reflection upon the nature of the primary tissue reactions which are noted in sinusitis. The products disseminated throughout the system from the nasal areas, once an acute local pathologic proce s is establi hed, are more or less toxic and their general nature is well understood. One of the main problems in sinus infections seems to be the restoration of that previous condition of the tissues which was characterized by *compatibility* with allergens or bacteria. The general nature of the biochemical changes converting *compatibility* into *incompatibility* is that of acidosis; this has been fairly established by the numerous biochemical studies of tissue changes in acute inflammation. The problems of acidosis are legion and are entirely aside from the subject-matter to be presented in this paper. Disregarding all details concerned with the acidotic process itself, antiacidotic measures are clearly indicated, among other therapeutic attempts to meet the general situation. This indication has been the backbone of the conservative treatments which have been applied in this series of cases.

Under "conservative" treatment is included certain *surgical measures*, namely: resection, synechia removal or reconstruction of septum; simple incision of antrum, bulla and sphenoid in giving vent to purulent content; removal of polyp, tonsillectomy and adenectomy.

*Nonsurgical Measures:*

*1.* Suitable regular exercise.

*2.* Dietary regime[3]: *a.* Elimination of foods known to disagree with patient. *b.* Selection of foods representing good vitamin bal-

ance. *c*. Selection of foods representing proteid, fat, carbohydrate balance. *d*. Fluid intake approximating 8 ounces per 10 pounds of body weight every 24 hours.

*3*. Periods of alkalization—as evidenced by neutrality or slight alkalinity of urine (alkalinity by no means precludes the possible existence of acidosis).

*4*. Periods of iodization—always controlled short of sharp focal reactions.

*5*. Use of salicylates sufficient to control pain.

*6*. Use of physiotherapy—by hot fomentations, radiotherapy or diathermy.

*7*. Local therapy—by tamponade (organic silver salts, or hygroscopic, or both)—negative pressure alone or in conjunction with tampons—medicated steam inhalations, with or without oil nebula.

*8*. Mechanotherapy—interdiction of hard nose-blowing, habituation to cleaning nose by suction down nasopharynx, negative pressure.

*9*. Avoid all nose washes—Dobell's, Seiler's, salt and soda, etc.; avoid insufflations of water in swimming; avoid diving and submerging the head in sea, pool or tub bathing.

*10*. Continue regime for months, regardless of prompt subsidence of symptoms; resume regime at intervals, even in absence of symptoms.

In special cases, endocrine imbalances indicate thyroid, insulin, pituitary or other suitable endocrine therapy; marked anemias require suitable additions in the form of drugs or diet; asthenia requires regulation of sleep and rest periods; overwork, worry, emotional or psychic disturbances, and any coincidental condition disturbing the general health, such as rickets, malta fever, malaria, tuberculosis or syphilis, must be controlled and eliminated as far as possible.

I wish to call special attention to the surprising value of regular, suitable exercise. I have had many patients declare their morning setting-up exercises, shower and rubdown do them more good than any local treatment. Of course, the details must be worked out for each individual respecting exercises and cold baths. Hot baths, foot baths, sweats and massage are also of definite value in many cases in conjunction with other measures.

In my experience a balanced diet, with adequate vitamin intake, especially A and D, is particularly needed by many patients. It is seldom sufficient to leave the selection of food to the patient. The pateint must be made to understand that the importance of proper diet does not extend over only a few days or weeks, but constitutes

an essential factor in the maintenance of health and freedom from nasal sinus disease.

Alkalization may be usually accomplished by diet. Some cases, however, are more satisfactorily alkalized by some such means as citrate and bicarbonate of soda, Wright's powder or citrocarbonate, taken with fluids three or four times a day.

Fluid intake should be largely between meals, usually at the rate of a glass an hour. The taking of a large fluid intake usually produces a decided antiacid effect.

Iodization must be approached cautiously; some cases can take only one dose at the beginning on account of lymphatic, skin and glandular focal reactions. Under supervision, iodide can be increased gradually without producing undue reaction until two or three doses per day are taken from three to five days at a time, producing a tolerable measure of rhinorrhea, with or without sense of activity in the parotid and cervical regions, perhaps also slight headache, abdominal discomfort or skin reactions. Most so-called "idiosyncrasies" subside under careful iodide administration.

Salicylates may be used with good advantage for control of pain and headache in connection with the treatment herein outlined. Aspirin in doses of one grain per 10 pounds of body weight, powdered up with 20 to 30 grains of bicarbonate of soda, followed by absolute rest, has proved satisfactory; this dose may be repeated if no amelioration of intense pain has been noted after 30 minutes. Morphin and opium should be avoided because of danger of addiction and of the undesirable antagonistic effects upon tissue reactionary processes. Atophan and phenacetin may be often combined advantageously with salicylate.

Physiotherapy in a form to stimulate rather than depress tissue reactions; in other words, to supply energy rather than abstract energy, has decided value as adjuvant measures in conjunction with the systemic attack outlined here. Heat is the most readily available form of energy for this purpose. Local therapy in the form of tamponade, negative pressure and medicated steam often proves of decided value, especially at the beginning of a course of systemic treatment.

The patient should be trained to avoid blowing the nose hard, learning to clean away tenacious secretion from the upper respiratory tract by drawing it backwards into the nasopharynx, to be expectorated.

The habitual use of alkaline or other nosewashes is to be discouraged, because of the undesirable effect of quick evaporation, which puts a stop to ciliary activity. Occasionally a nasal douche may be

necessary, but it is always succeeded by a period of dessication of the cilia, during which secretion lies unmoved, evaporating into films and crusts, which again demand lavage for their removal. The same is true of water insufflated while swimming or bathing. Cocainization produces paralysis of ciliary activity, a particularly undesirable condition of the affected nasal areas.

Most important is to impress upon the patient at the start that this general regime is to be kept up, more or less as outlined, for months or years, regardless of improvement.

I have had opportunity to observe the condition of patients over periods of years under the influence of such a general regime as has been outlined; in comparison with similar cases treated by radical surgery or by local nasal treatments, such as antral lavage, cautery, tampons and topical applications, the results of the measures herein advocated are definitely superior. The incidence of serious complications, such as osteomyelitis, meningitis, brain abscess, thrombosis and septicemia, in my experience has fallen to *nil;* during the past ten years I have not seen one case so treated develop intracranial complication. I have seen such complications in many cases during this time; they were, without exception, postoperative complications. While I am prepared to believe that such complications may arise without previous surgery, I have not seen one in ten years. Regardless of what *might* happen in this respect, it is *certain* that they are much more apt to occur under the conditions usually obtaining in the average course of radical nasal surgery.

*Summing up:* Upper respiratory conditions alluded to in this paper are made one with the general systemic conditions under which they develop. Co-operation on the part of general systemic mechanisms of resistance and recovery are solicited to a maximal degree in coping with disease conditions. The essential upper respiratory tissues are to be given all possible opportunities to regain normal condition, and to conserve functionability to the utmost, not only for present use, but for withstanding similar vicissitudes of the future. The upper respiratory tissues constitute an important part of the general mechanism of immunization and, as such, their preservation has important bearing upon the general health of the individual.

BIBLIOGRAPHY.

1. WRIGHT: Annals O. R., and L., March, 1928.
2. PINESS: Personal Communication.
3. Since presenting this paper, Jarvis has submitted his excellent "The Upper Respiratory Tract and Guide to Nutritional Disasters," read Jan. 4 before the Eastern Section, A. L., R. and O. His conclusions are of great value and should be given studious consideration.

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# A CONSIDERATION OF PERITONSILLAR ABSCESS WITH A RATIONAL SURGICAL METHOD OF RELIEF.

Dr. O. R. Dobbs, St. Louis.

Peritonsillar abscess, quinsy, cynanche, is a condition that is familiar to every otolaryngologist, as well as to every general practitioner of medicine, and as familiarity often breeds contempt, the peritonsillar abscess has become contemptible. In the individual affected, however, a deep and lasting respect is rapidly created. Few things become so uncomfortable and the relief surgically as a whole so painful. Harry Barnes[1] speaks of it as the most uncomfortable surgical condition with which we have to deal. H. L. Baum[2], an advocate of removal of the tonsil during an attack, believes that the pain incident to tonsillectomy is a relief to the pain attending a peritonsillar abscess. Usually the patient recovers from an attack of peritonsillar abscess but, unfortunately, either directly or indirectly, death may result. A resumé of the literature on this subject reveals a surprisingly large number of fatalities when we consider that the condition is one that ordinarily would not be reported.

Historically*, the disease is an old one and the treatment, to a great extent, has remained about the same for many centuries. We first see this description in Egyptian medicine in the "Papyros Ebers", the first of all medical writings, "If thou findest in the throat a tumor and it appears like an abscess of the flesh which can be reached by the fingers, thou must say thereto, I will treat the disease with a knife, taking care of the vessels."

Hippocrites applied the term "cynanche" to all acute inflammations of the throat and the cause was supposed to be a coagulation of the blood in the neck. Celsus, at about the beginning of the Christian era, makes a further division. He states that the Romans called any inflammation which was confined to the fauces, "Angina", while the Greeks gave the term "Cynanche" to that form causing dyspnea without the appearance of inflammation of the fauces. Galen later used the same classification.

Over a thousand years later, the Italians, in the School of Salermo had translated Cynanche into Squinantia. The Greek Cynanchia had thus emerged into the medieval Latin Squinantia, and in

*A History of Laryngology and Rhinology; Jonathan Wright.

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the eighteenth century in English to "Squinsy" in the writing of Huxam, and at the present time to Quinsy.

It is from the teaching of Celsus that we have our present method of opening a peritonsillar abscess. He made deep incision into the palate above the uvula. He also made deep incisions externally beneath the jaws and bled from the lingual veins; and prescribed the burnt ashes of a swallow sprinkled on the part locally. Pliny and Galen speak highly of the efficiency of this prescription, even differentiating on the effect of different kinds of swallows.

Thirteen hundred years later we quote from Gui di Cauliac, the great surgeon of the middle ages, "The abscess having matured, first incise it with a lancet if it is to be seen, and the mouth rinsed out with parsley. If, however, it is so far within as not to be seen, it should be broken with the fingernail or by rubbing with something if possible." He also quotes the following ingenious method of treatment for quinsy: A half-cooked piece of meat should be taken and tied to a long, strong cord and the patient be made to swallow it, and while he was swallowing it, it should suddenly be jerked out with violence by the cord and the abscess thus ruptured. The same may be done with a sponge.

This seems to be an application of the methods of Aeteus, of Constantinople, in the sixth century for removal of foreign bodies from the throat. This method of removal of foreign bodies was carried on by Damascenus, the Arab, a follower of Albucosi. This writer also gives an interesting method of cure of any inflammation about the palate: "This method of cure is the diversion of the cause and performed in a manner which causes the trouble to shift its seat, in short, rubbing of the ears and pulling them forcibly upward, and the painful stretching of them and the application of cups to the opposite parts for these things raise the inflammation and bear it upward, and among those things useful in the elevation of it is that a handful of hair should be grasped in the hand and the patient told to keep silent, then put thy feet on his shoulders and drag strongly on the handful of hair until the skin is pulled off, for by such dislocation will pharyngitis be raised."

The patient with an advanced peritonsillar abscess presents a definite and familiar appearance—facial expression of impending disaster—marked distress—head thrust forward on the shoulders and carried slightly to the affected side—mouth slightly open, keeping the distance between the teeth stationary and, when trying to speak, using the tongue and lips as little as possible. Any attempt to open the mouth or depress the tongue is met with opposition and

the use of a local anesthetic, either for examination or treatment, is unavailing to any great extent, as the pain occasioned by any manipulation is in direct ratio to the amount of pressure applied. When using a blunt instrument, such as a forceps or scissors, there is considerable pressure necessary to plunge it into the palate to a depth sufficient to reach the abscess. It is an operation that is painful to the extreme, and I have frequently lacked the stout heart necessary to use a sharp bistoury for the same purpose, especially when my perspective is limited because of inability to see clearly.

The method I have used during the past two years is to open directly into the supratonsillar fossae. This method, of course, is applicable only when the peritonsillar abscess is a supratonsillar one; however, 90 to 95 per cent of all quinsies are supratonsillar.

In attempting to open into the supratonsillar fossa, the superior border of the anterior pillar, where it merges into the palate, is followed as a guide and while this border is not so evident in a healthy throat, it is present as a well defined fold in a swollen palate. In fact, it seems to be placed there for this particular purpose. In following the fold of the anterior pillar, the usual anatomical relationship has been changed. Normally, the anterior pillar presents a slight and gradual curve, which blends abruptly into the palate. In a peritonsillar abscess there is pressure behind the tonsil, and in the soft palate; the tonsil is more adherent and more closely applied to the posterior than to the anterior pillar. The tonsil, therefore, is not only protruded into the throat, but rotated as well. This tends to make the anterior pillar more of a straight line. The pressure, plus the swelling of the palate, quite naturally extends the palate downward and forward, forming a shelf-like projection above the tonsil and bringing the upper part of the anterior pillar into bold relief. This was always present in the cases I have seen and is the guide to the supratonsillar fossae. With this method of opening a peritonsillar abscess, a series of probes are used, the first having been a small blunt end; this is followed by a larger instrument, which is threaded. The threaded instrument readily takes hold as it is screwed in, following the smaller opening already made. Very little pressure is necessary to enter the cavity and when the instrument is abruptly withdrawn leaves a larger opening than one made by a smooth instrument. The opening can then be enlarged as required, using a larger probe or a small forcep.

By following the fold of the anterior pillar as described, the thin membrane, which is the only barrier to the supratonsillar fossae, is easily found and very little pressure is then necessary to penetrate

this membrane. The instrument is felt to enter the cavity with much the same assurance and ease as in entering the spinal canal when doing a spinal puncture. This is done without any anesthesia, practically without pain and with very little bleeding. In some cases it will probably be necessary to repeat the procedure; however, because of the relief afforded and the ease of manipulation, the patient is quite willing to have it done. More difficulty is encountered in recurrent attacks of peritonsillar abscess because of scar tissue formation. This method, I believe, can be done several hours earlier, in many cases, than by opening through the palate. Ordinarily, a peritonsillar abscess is allowed to thoroughly ripen before attempting to open it. This increases the danger of sepsis and hemorrhage, besides trying the endurance of the patient.

In one case treated early, a small amount of serum was evacuated and the condition subsided. Should there be a mistake in diagnosis, relatively little harm has been done, because of the small amount of trauma, and no open wound left to slough.

The efficiency of this method is dependent for the most part on two factors: *1*. The site of approach—being the most natural, both mechanically and anatomically. *2*. By using a series of instruments so constructed as to form a sufficiently large opening with a minimum amount of trauma.†

## SUMMARY.

*1*. The present-day method of surgery in peritonsillar abscess is unsatisfactory both to the patient and surgeon.

*2*. The method described is simple, easy of application, and is the most natural way of approach.

*3*. It is relatively free from pain and danger to the patient.

*4*. It lessens scar tissue formation, thus making further surgery more gratifying, either in recurrent attacks of peritonsillar abscess or tonsillectomy.

*5*. Earlier application, possibly saving the patient a number of uncomfortable hours and lessening danger of sepsis, extension of the condition, and hemorrhage.

*6*. Trauma is reduced to a minimum, thereby obviating further swelling and extension should the attempt prove unsuccessful. It leaves no open, sloughing wound, in case of systemic diseases such

†See subsequent article by Dr. M. D. Pelz.

as diabetes, and, lastly, very little harm is done, even though there should be an error in diagnosis.

REFERENCES.

1. BARNES, HARRY A.: Trans. Amer. Laryngological Assn., June, 1915, p. 368.

2. BAUM, H. L.: Radical Cure of Peritonsillar Abscess. Ann. Otolaryngology, Rhinology and Laryngology, 35, pp. 429-437, June, 1926.

2435 N. Grand Boulevard.

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## A NEW INSTRUMENT FOR EVACUATING PERITONSILLAR ABSCESSES.

DR. MORT D. PELZ, St. Louis.

The instrument herewith presented has been used successfully by the writer in opening peritonsillar abscesses, without the pain and bleeding associated with the classical method of incision or puncture into the soft palate. As almost all peritonsillar abscesses occurring for the first time, and more than 90 per cent of all types, including subsequent occurrences, are evacuated through an opening into the supratonsillar space, the writer has followed the method described


Fig. 1.

by Dr. O. R. Dobbs in the preceding article (in this journal) and has used the natural cleft beneath the plica semilunaris as the site of evacuation.

The instrument consists of a graduated screw, blunt at the tip, with a swivel head at the opposite end. The swivel head is similar to that of a watchmaker's screw driver (see Fig. 1).

In using the instrument it is held almost horizontally, and the tip is applied under the plica semilunaris at a point where the anterior pillar merges into the soft palate. It is directed upwards toward

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the supratonsillar space. A few rotations of the swivel end of the instrument will cause the blunt-ended tip to enter the abscess cavity; the pus being evacuated through this opening. A closed hemostat may then be introduced and gently spread apart, for the purpose of increasing the size of the opening; generally this procedure is not necessary. By this method the writer has drained peritonsillar abscesses with no bleeding or pain.

A further advantage of this method is that in many cases where the mouth cannot be opened sufficiently for incision, this instrument can be placed in the proper position and while the patient's mouth is almost closed the screw is introduced into the abscess cavity.

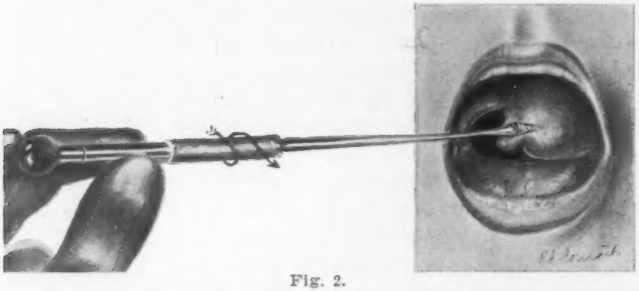

Fig. 2.

Occasionally incisions have been made for the evacuation of peritonsillar abscesses before pus had sufficiently localized or while the condition was a peritonsillitis rather than a peritonsillar abscess. Such ill-timed incisions may result in severe complications, including cellulitis and suppurations in the retro- and parapharyngeal spaces. The writer believes that many such complications can be prevented by this method, because the trauma is minimal and there is no counter-opening through noninfected tissue as is the case with the classical incision.

634 N. Grand Boulevard.

# REPORT OF A CASE OF FOCAL INFECTION NEUROLABYRINTHITIS MANIFESTING NYSTAGMUS OF A CHARACTER ASSUMED TO BE OF PURELY CEREBELLAR TUMOR ORIGIN.*

DR. GEORGE W. MACKENZIE, Philadelphia.

The character of the nystagmus present during the height of the involvement was so different from that usually found in neurolabyrinthitis as to afford in itself ample reason for reporting the case. When we take into account also the fact that the nystagmus was occasionally of a form recognized by such leading authorities as Marburg[1], Spiegel[2], Leidler[3], Brunner[4] and others as typically of cerebellar tumor origin, there is additional reason for reporting it.

The case, Dr. F. L. G., age 71 years, was referred by Dr. Leroy I. Walker, of Philadelphia. The patient was first seen by the writer on the evening of April 20, 1929, at his home, where he was confined to his bed. He claimed that on last Thursday morning (April 18), when he attempted to get up out of bed the room seemed to go *up and down*. Questioning the patient more closely as to the direction of the apparent movement of the room, he repeated emphatically that it was up and down and not horizontal nor rotary (frontal). He went back to bed and asked his wife to call in Dr. Walker. During the examination that followed Dr. Walker thought he recognized a "slight congestion of the right ear." The patient said he could not move his head without producing vertigo and pain in the front of the head. He denies ever having had a discharge from either ear and claims to have always had keen hearing. Mrs. G., who was standing by, remarked that the patient's hearing seems to her to be slightly impaired since the attack of the vertigo. Dr. Walker suggested calling in the writer for a more detailed study of the case.

At this first visit the patient felt satisfied that his vertigo had improved considerably in the last two days, but not sufficiently to allow him to be up and out of bed. He was treated expectantly until sufficiently improved to report to the office for closer study.

*Otoscopic examination* reveals both drumheads to be normal; that is, they were intact, brilliant, normally translucent, showing no

*Read before the Philadelphia Laryngological Society, Dec. 3, 1929.
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bulging nor retraction. With the Siegel instrument they move normally, likewise with Politzer inflation.

May 14, 1929: As the patient came into the examining room to sit down, he made a slight movement of the head forward and then backward (the usual compensatory movements that we all make in taking a seat in a chair), he stopped suddenly and held himself rigid because of a sudden attack of vertigo. Examining the eyes during the attack, with the patient looking straight ahead at a distant object across the room, revealed the presence of a very intense mixed rotary-horizontal nystagmus to the right side. It lasted for about two minutes and then stopped. This was followed by a nystagmus downward that was almost questionable.

On having the patient repeat experimentally these head movements, that is, one slight movement forward and one backward, no vertigo was experienced nor was the intense nystagmus repeated; but, when the head was moved as far as 90° forward, that is, with the face directed toward the floor, the patient experienced severe dizziness, which gradually eased up, finally disappearing after a few minutes. During this dizzy attack no attempt was made to examine the eyes for nystagmus, but later this was done.

Moving the head backward 90° (face directed toward the ceiling) produced a mixed rotary-horizontal nystagmus toward the right side. Incidentally, he complained of noises in the left ear, which he had had for several years. He was then directed to lean forward, facing the floor, when there developed a horizontal nystagmus directed toward the left side. After three or four seconds the head was brought to the normally erect position, when the nystagmus changed to mixed rotary-horizontal to the right side, with the horizontal element predominating. These experiments were repeated several times for the purpose of checking up with the first observations, and always with the same results. It might be added that the length of the excursions at the equator of the globe was between 2 and 3 m.m.

According to the patient his blood pressure is 145 systolic; the diastolic was not recalled. He claims that his "urine is negative."*

The urinalysis to which the patient refers was made by Dr. George A. Hopp. It reads as follows: Appearance clear; color, amber; reaction, acid; specific gravity, 1.020; albumin negative; sugar negative; acetone negative; bile negative; indican absent; urea, 2 per

*The records of the case have not been rearranged in order to present them in the best shape; they are presented in their chronological order for the sake of greater accuracy. It is next to impossible to make a complete examination of a case suffering from vertigo in one visit, especially in a patient 71 years of age.

cent. *Microscopic:* Casts negative; pus negative; blood negative; epithelium present; crystals negative. *The Blood Count:* Hemoblobin, 75 per cent (Dare); leukocytes, 8,000; red cells, 3,800,000; polynuclears, 61 per cent; lymphocytes, 36 per cent; transitionals, 2 per cent; eosinophiles, 1 per cent. Red cells are regular in shape and size. (Signed) George A. Hopp.

X-ray study of the teeth, made April 22, 1929, reads as follows: "Roentgenologic study of the entire denture reveals evidence of the following: The upper left remaining molar, the upper left cuspid, the lower right second bicuspid, the lower left cuspid and the first bicuspid all show well defined evidence of chronic periapical pathology. The upper right lateral incisor shows a marked degree of pericemental pathology and appears to be a necrotic tooth. There is a small fragment of root in the region of the lower left first molar. The alveolar process shows evidence of pyorrhetic resorption, with infiltration of the peridental membranes in the lower denture. (Signed) Joseph W. Post."

May 23, 1929: The patient reports having had some teeth removed since his last visit and as a result feels like a "fighting cock." He says that he is convinced that the removal of the lower right second bicuspid brought about his marked general improvement. In fact, he felt so much improved that he went out and worked in the garden, planting flowers. He has had very little vertigo since his last visit; however, he felt somewhat dizzy this morning when stooping over, lacing his shoes.

Examination for spontaneous nystagmus in the side positions for comparison is rather difficult to make since the right eye is blind, from a nearly ripe cataract. For this reason he cannot look so far to the right side as the left. Any normal individual, that is, one free of any vestibular vertigo, under such a visual handicap, will manifest a more pronounced nystagmus to the left than to the right side, for the simple reason that it is possible to look more strongly toward the side corresponding to the seeing eye than toward the side of the blind eye.

At this visit (May 23) there was observed a vertical nystagmus downward with the patient looking straight head at infinity, confirming our observation of vertical nystagmus downward at the time of the first visit, when its character was somewhat obscured by the coarser lateral and rotary movements of the eyes, produced by shifting the position of the head.

The nystagmus on this day was repeatedly studied by Dr. W. G. Shemeley and myself. Its direction was carefully noted, the length

of excursions estimated and its frequency timed. Its direction was vertically downward, length of excursion somewhere between 1 and 2 m.m., its frequency averaged five movements in 20 seconds. When the patient moved the head downward 90°, neither its character nor frequency changed as it did on the previous visit. With the face directed toward the ceiling there was a horizontal nystagmus toward the left side, combined with vertical nystagmus downward. In other words, there was an oblique nystagmus downward and to the left; 27 excursions in 22 seconds, with an average amplitude of 1½ m.m.

GALVANIC TEST:

| *Right Ear* | *Left Ear* |
|---|---|
| Kathode 6½ ma. produces a mixed rotary-horizontal nystagmus to the right. | Kathode 6 ma. produces a mixed rotary-horizontal nystagmus to the left. |
| Anode 6½ ma. produces a mixed rotary-horizontal nystagmust to the left. | Anode 7 ma. produces a mixed rotary-horizontal nystagmus to the right. |

FUNCTIONAL HEARING TEST:

| *Right Ear* | | *Left Ear* |
|---|---|---|
| | < Weber | |
| norm. | Schwabach | norm. |
| +35″ | Rinne | +35″ |
| norm. | Air | norm. |
| norm. | $C_1$ | norm. |
| norm. | $c_4$ | short 5″ |

June 1, 1929: The patient reports that the vertigo has been improving right along, and much more so since the removal of a portion of the lower left molar tooth a week ago. While he was having vertigo he stopped smoking, but in the last few days he has been smoking again, with no bad results. He claims to feel better generally and stronger and his muscles feel hard.

Hearing tests for the low, middle and high tones made on each ear separately reveal the hearing to be normal. At the conclusion of the hearing test he reminds us of the fact that his hearing has been very poor in the left ear prior to his first visit to this office.*

*Spontaneous Nystagmus:* When looking straight ahead, there is a questionable nystagmus to the left and somewhat downward. Violent movements of the head downward and forward and, in fact, in all directions do not produce vertigo nor increase the existing nystagmus; whereas, before the extraction of the teeth the slightest movement produced intense vertigo with nystagmus.

*This is a contradiction of the original history he gave us, when he claimed that he "always had keen hearing."

June 11, 1929: The patient reports that he has had "no vertigo to speak of." Occasionally, he has a slight buzzing sound in the head. He claims, too, that his general condition is markedly improved.

Spontaneous nystagmus in the lateral position of the eyes is difficult to compare, owing to the blindness in the right eye referred to elsewhere. When he looks to the left side there is an oblique nystagmus downward and to the left, of very slight degree. On looking straight ahead there is no nystagmus present. Neither is there any nystagmus when looking upward.

FUNCTIONAL HEARING TESTS:

| *Right Ear* | | *Left Ear* |
|---|---|---|
| | Weber | not lateralized |
| norm. | Schwabach | norm. |
| +38″ | Rinne | +38″ |
| norm. | Air | norm. |
| norm. | $C_1$ | norm. |
| norm. | $c_4$ | norm. |

Nov. 21, 1929: The patient reports that he has been feeling well for the past few months. At the present time he has no headache, no dizziness and feels well in all respects. Appetite has greatly improved; in fact, so much so that he finds it "necessary to ease up in eating." He has gained nine pounds in weight since the last visit in June.

Functional hearing tests reveal the same normal findings as on June 11, 1929.

*Examination for spontaneous nystagmus* reveals no evidence of nystagmus when looking straight ahead and only the normal physiological nystagmus when looking to the right and left sides.

SUMMARY OF THE CASE REPORT.

That the cause of this patient's vertigo was due to the presence of a focus of infection in the alveolar process is established by the following facts:

*1.* That vertigo is commonly the result of a focus of infection. According to the experience of the writer it is by far the most frequent cause. In most European countries, where focal infection is referred to rather slightingly as the "American idea," they prefer the use of the less definite term, "toxic."

*2.* That the site of the focus of infection responsible for neurolabyrinthitis is most frequently found in the alveolar process.

*3.* That this case behaved in the main like other cases of known focal infection origin would prompt us to include it in the same group.

*4.* That the removal of the focus of infection in the alveolar process resulted in a prompt clearing up of the vertigo and its accompanying sign, nystagmus.

*5.* That along with the improvement in the vertigo there was such an improvement in the patient's general condition as to prompt him to report that he felt like a "fighting cock" and that he had been out in the garden planting flowers; and, still better, that he had gained nine pounds in weight.

*6.* That no other form of treatment was used than the extraction of teeth.

On the other hand, the nystagmus was not altogeher typical for neurolabyrinthitis of focal infection origin. In most of the cases of dental origin studied by the writer, the involvement was unilateral when the nystagmus was more often directed away from the affected side and was of the mixed rotary-horizontal form. The writer in the study of many cases had never before observed one in which there was any vertical element. That there was a vertical nystagmus is undeniable. Three careful observers, Dr. Wm. G. Shemeley, Dr. Alice V. Mackenzie and the writer, all saw it at the same time and again at different times. Furthermore, the patient's first report of his vertigo was that of a sensation of the room moving up and down, and when questioned he repeatedly adhered to his original statement, denying that the room moved in some other direction, confirming in a subjective way the plane of the nystagmus.

Another outstanding symptom was the production of nystagmus by *vertical* movements of the head when he sat down in the chair on the occasion of his first visit to the office. Again, when he would stoop forward, facing the floor, experimentally at the office and when lacing his shoes in the morning, vertigo was induced. Unfortunately, nystagmus was not looked for with the first severe attack at the office. It came so suddenly that it caught us unprepared; but later it was looked for and found. On May 23 three of us observed the nystagmus repeatedly and sufficiently often as to cause us to feel sure of the following facts:

*1.* That the patient in the beginning of his vertigo saw the outside world move in the *vertical plane,* and no other plane.

*2.* That a quick movement of the head in the *vertical plane,* and no other plane, precipitated attacks of vertigo; furthermore, that it was a movement *forward* and not backward that caused the attacks.

*3.* That *vertical nystagmus* was observed during the experimentally induced attacks of vertigo, and to a lesser degree between the attacks.

On the other hand, it cannot be denied that the patient also manifested nystagmus in other planes, for we found:

*A.* That on more than one occasion on raising the head from the 90° forward position (face directed toward the floor) to the erect position, caused the nystagmus to change to a mixed form of rotary-horizontal to the right. The same as one would expect to find in the case of unilateral left-sided neurolabyrinthitis of the focal infection form.

*B.* That inclining the head backward caused the nystagmus to change to a combined horizontally left-downward; in other words, an oblique nystagmus downward and to the left. On some occasions the horizontal element predominated, and at other times the vertically downward. Variability in the intensity of the nystagmus, and perhaps, too, in its direction, at different times is explainable on the basis of differences in the amounts of toxins in the system. For instance, those days in which the elimination was freer the symptoms and signs of toxemia, including vertigo and nystagmus were less pronounced, and *vice versa.* Again, following each extraction there was a diminution of toxins generated at the source and with it a corresponding improvement clinically. This improvement manifested itself in a lessening in the intensity of the vertigo and a reduction of the horizontal and rotary elements of the nystagmus, allowing the vertical to stand out in relief.

Why any case of focal infection neurolabyrinthitis should manifest vertical nystagmus with its associated subjective sensation of motion in the vertical plane in contradistinction to that found in every other case seen by the writer, is unexplainable at this time. If by any stretch of the imagination we could assign the site of the lesion responsible for the vertigo to any other place than the inner ear or nerve, we could not so readily deny the etiological connection of the focal infection; in other words, the source of the toxemia in the alveolar process.

The presence of mixed rotary-horizontal nystagmus on some occasions, for short periods, and vertical and oblique nystagmus on other occasions, for longer periods, naturally invites a discussion of the generally accepted beliefs as to the etiology of each form.

Spontaneous rhythmic nystagmus can be produced by a lesion in the inner ear or the vestibular branch of the eighth nerve when it is designated peripheral nystagmus. It may be produced also by a lesion more centralward; that is, in the nuclei or their tracts, when it is designated central nystagmus. Peripheral and central nystagmus present definite characteristics which make their differentiation possible. They are as follows:

PERIPHERAL NYSTAGMUS.

1. The *plane* of the spontaneous eye movements may be horizontal, rotary or mixed rotary-horizontal. A horizontal nystagmus would indicate a circumscribed lesion in the inner ear (the horizontal semicircular canal). A purely rotary nystagmus would indicate a circumscribed lesion in one of the vertical canals. A mixed rotary-horizontal nystagmus, on the other hand, speaks for a diffuse lesion of the inner ear or of nerve involvement. A single plane nystagmus from a nerve lesion is quite inconceivable. Multiple plane eye movements is by far more common than the single plane.

2. The *direction* of the nystagmus indicated by the quick movement of the eyes in the case of a very mild lesion (simple congestion of the inner ear) is toward the same side; in the more severe lesions, those constituting the vast majority, the nystagmus is toward the sound side. If one inner ear is destroyed, there occurs immediately a mixed rotary-horizontal nystagmus directed toward the sound side.

3. *Duration* of the nystagmus in a destructive lesion is brief. It is very intense at the beginning but rapidly diminishes. At the expiration of a month or so it is hardly noticeable, except to the most carefully trained observer. Compensation appears to take place early, as in the case of animals upon which the experiments of extirpation has been done (Bechterews compensations).

4. The *subjective symptoms* (vertigo) are very pronounced at the start and gradually lessen so that by the end of four or five days they become tolerable. At first the vertigo is so intense that the patient seeks a recumbent position and remains as quiet as possible. His symptoms are aggravated by the slightest motion of the head. His nausea is intense and he vomits with it. There are also present, in recent cases, pronounced symptoms referable to the vegetative system.

CENTRAL NYSTAGMUS.

1. The *plane* of the eye movements except in a very extensive lesion is generally single. In other words, the nystagmus occurs in either the horizontal, the frontal, or the vertical plane in contrast to the multiple planes nystagmus (mixed rotary-horizontal) so common in the peripheral form. Vertical nystagmus is characteristic of the central form; so, too, is the oblique. Oblique nystagmus is much less often seen than the purely vertical.

*2.* The *direction* of the nystagmus is usually toward the sound side. A destructive lesion in the caudal part of Deiter's nucleus produces nystagmus toward the side of the lesion; whereas, one in the cranial (oral) part produces a nystagmus toward the opposite side. If the nystagmus is toward the side of the ear lesion, we would think of a central lesion, the more so if the labyrinthine reactions were negative.

In the case of central lesions we frequently find other symptoms referable to the brainstem. For instance, anesthesia in the distribution of the fifth nerve. Sometimes involvement of the tenth nerve, as in the case of the Fremel syndrome.

*3. Duration* of the nystagmus, unless something intervenes to modify it, seems to be drawn out indefinitely. The intensity does not appear to lessen, as in the case of a peripheral lesion.

*4.* The *subjective symptoms* (vertigo) are not so pronounced. In some cases there is practically no unpleasant subjective sensation, in spite of the wide excursions made by the eyes. Besides, there is an absence of symptoms referable to the vegetative system.

Marburg is credited by Brunner with having been the first to find horizontal nystagmus in lesions of the ventrocaudal part of Deiter's nucleus; whereas, vertical nystagmus with the quick component directed upward was found in the case of a cysticercus extending from the region of the abducens nucleus above to Roller's glossopharyngeal root below. The clinical observations of Marburg prompted Rudolph Leidler to make some experimental investigations on rabbits. The result of his experiments led him to the following conclusions: Spontaneous horizontal nystagmus prevails in lesions reaching from the caudal beginning of Deiter's nucleus to the knee of the facial; rotary nystagmus in lesions of the most caudal part of this region; vertical nystagmus in the lesions of the oral part of Deiter's nucleus.

Concerning the cerebellar origin of nystagmus, there seems to be some doubt as to whether a cerebellar lesion produces nystagmus directly or indirectly. By indirectly is meant its "distance working" effect. Brunner*, who has written quite extensively on the subject in recent years, in one place refers to the etiology of central nystagmus as arising from diseases in: *1.* The brainstem; *2.* the cerebellum; *3.* the cerebrum. Elsewhere he emphatically states that the "cerebellum does not produce nystagmus." This contradiction is

*Brunner in a footnote on this subject states that rotary nystagmus in rabbits corresponds to vertical nystagmus in the human, and vice versa.

more apparent than real; for cerebellar disease in itself may not be responsible for nystagmus. At the same time a tumor or abscess of the cerebellum may cause the enlarged cerebellum to press upon the brainstem and produce a central nystagmus, or it may so crowd the region of the eighth nerve about the internal auditory canal as to produce a peripheral nystagmus. There remains one case, however, seen by the writer with the late Dr. W. H. Sears, of Huntingdon, Pa., that is not so easily explained on this basis. Briefly, it was one of labyrinthine suppuration, upon which we operated, where the patient manifested a mixed rotary-horizontal nystagmus toward the side opposite the dead labyrinth. At the completion of the labyrinth extirpation and while the patient was still under the general anesthetic, the cerebellum on the same side was incised for a supposed abscess, which was not found. Following the operation the patient manifested a very wide excursion, horizontal nystagmus directed toward the operated side, without vertigo. This case was incompletely reported before the American Laryngological, Rhinological and Otological Society in 1927, referred to as Case 11 in the series of Suppurative Labyrinthitis. This observation, though it was made by the writer 14 years ago, has been recently confirmed by Brunner. In bold print he says, "as a rule, labyrinthine nystagmus, insofar as it is coarse, or of high intensity without intense vertigo, is not a peripheral but a central labyrinthine nystagmus."

According to Spiegel, there is one form of nystagmus that is produced by the indirect effect of a tumor of the cerebellum; that is, when the tumor of the cerebellum is situated medianly and forward. In this case the patient in the upright position will often be quite free of nystagmus and yet when he leans forward there develops nystagmus because the tumor has a better opportunity of weighing down on the Corpora quadrigemina in the neighborhood of which is found that area which is responsible for vertical nystagmus.

The case I have just reported presented the following signs suggestive of a central lesion: *a*. Verical nystagmus; *b*. oblique nystagmus; *c*. aggravation of symptoms (nystagmus and vertigo) when leaning forward. This last symptom is characteristic of central nystagmus produced indirectly by a tumor of the cerebellum, according to Spiegel.

In conclusion, the writer wishes to emphasize two points: *1*. His belief that the case was one of focal infection neurolabyrinthitis. *2*. That in view of the similarity of the symptoms in this case to those of cerebellar tumor, so far as the nystagmus is concerned,

prompts him to the conclusion that one is not warranted in feeling too "cock sure" about pathognomonic or even characteristic signs in diseases of the nervous system.

BIBLIOGRAPHY.

1. MARBURG, OTTO: Alexander and Marburg Handbuch der Neurologie des Ohres. Band 1, S. 992.

2. SPIEGEL, E. A.: Recent Lectures on Neuropathology to American Students in Vienna. 1927-28-29.

3. LEIDLER, RUD.: Experimentelle Untersuchungen ueber das Endigunsgebiet des Nervus vestibularis. 1. Mitteilung: A and B a. d. neurolog. Institute Wien, 1913, 20, 256. 2. Mitteilung: A and B a. d. neurolog. Institute Wien, 1914, 21, 151. Also quoted by Brunner in Alexander and Marburg Handb. d. Neurolog. des Ohres.

LEIDLER, RUD.: Ueber die Beziehungen der Mult. Skler. z. centralen Vestibular. Monatschr. f. Ohr., 1917, 51, Jahrg.

LEIDLER, RUD.: Ueber die Beziehungen der Syringo myelie (resp. Syringo bulbie) zum centralen vestibular Appar. Zeitsch. f. Ohr., 1918, 76, 201.

4. BRUNNER, HANS: Alexander and Marburg, Band. 1, p. 1001, in bold type, claims that as a rule a labyrinthine nystagmus when of wide excursions or of high intensity without vertigo is not peripheral; but of central labyrinthine origin. This was a well recognized fact as long ago as 1908.

1724 Spruce Street.

# THE ROLE OF THE EOSINOPHILE IN CERTAIN OTOLOGICAL CONDITIONS.*

DR. CHAS. C. W. JUDD, Baltimore.

For me, a mere practitioner of medicine, there has been a very justifiable hesitancy to address so specialized a group as you oto-laryngologists on any matter pertaining to your practice.

My timidity was overcome in the first instance by the signal honor tendered me by your Society's invitation to address you on this occasion. To this very considerable reason was added another: Not once, but repeatedly, I have observed in cases of otitis media and its sequels a fairly constant and distinctive blood picture, to which I have grown to attach a high degree of significance. It is one which, so far as I can find, has not, up to the time of my stumbling upon it, been emphasized. Should an exposition of this phenomenon in any way aid my Philadelphia confreres, I shall indeed feel doubly grateful for this opportunity of appearing before you.

You probably once knew, and, with excuse, have doubtless since forgotten, that the eosinophilic cells of the circulating blood are increased with great regularity as a result of the parenteral entrance of alien protein. You will possibly recall as examples of this phenomenon the tremendous increase in these cells occasioned by the invasion of our tissues by trichinellae and the less marked eosinophilia induced by other nemetodes whose invasion of our bodies is confined to the intestinal lumen and its walls. In your own field, you are all familiar with the fact that, in pollen allergy (again because of the absorption of an alien protein) there is usually an associated eosinophilia. In like manner in other allergic diseases, as bronchial asthma, urticaria and serum sickness, the introduction of the very alien protein which precipitates the attack also gives rise to an absolute increase of the oxyphilic granular cells of the blood-stream. In most of these conditions the total leukocyte count is not materially altered from the normal.

In contrast with this picture, most bacterial infections, and especially those due to pyogenic bacteria, induce a decided increase in the total leukocyte count with an attendant *hypo*eosinophilia and a more or less pronounced increase in the neutrophilic granular leukocytes. To this combination of leukocytosis, hyperneutrophilia and

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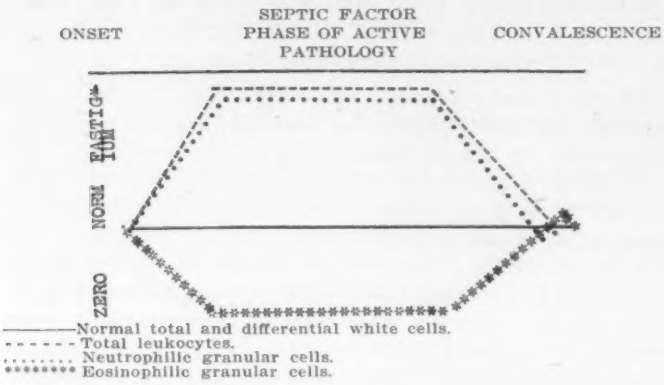
hypoeosinophilia, the name of *"septic factor"* has quite appropriately been applied.

DIAGRAM I.

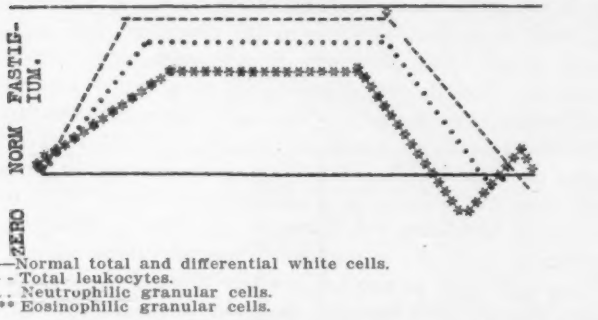



Still another type of blood picture, involving changes, chiefly in the granular cells, is found in conditions where extensive autolysis of tissue is taking place within the animal body. Massive tubercles, large gummata and neoplasms undergoing necrosis are examples of the conditions producing this change. Here there is a high total leukocyte count, a marked neutrophilia and the eosinophiles are in normal or even increased numbers. Rather hesitatingly, and yet not without some justification, I have been applying the term *"lytic factor"* to this phenomenon. In its production we are convinced that two processes are operative in the same lesion. The first stimulus is furnished by the absorption of alien protein derived from the autolysate of the broken-down tissue, giving rise to a hypereosinophilia, and the second stimulus is due to a secondary invasion of this necrotic tissue by pyogenic organisms. The composite picture of a hypereosinophilia and a "septic factor" yields the graph of a "lytic factor."

With these considerations in mind, let us turn our attention to your field of practice and consider the common sequence of events in acute otitis media. Commonly, as an extension from the nasopharynx, the middle ear is involved in an acute pyogenic infection. The serous type of otitis usually subsides in the course of a few days, the purulent type lasts somewhat longer. When the inflammatory area does not extend beyond the limits of the middle ear, the pain, fever and discharge gradually diminish, to disappear in a week or ten days.

I hope that most of you will agree that when a purulent otitis persists over two weeks, the burden of proof rests upon him who maintains that there is no extension of the inflammatory area to the attic and the mastoid cells. It does not follow that all of these extensions require a mastoid operation. Indeed, I am convinced that there is a type of low-grade infection involving some of the mastoid cells in which operation is quite unnecessary and should not be contemplated as the tendency is toward spontaneous recovery. Even in the development of extensive mastoiditis, I am emphatically convinced that there is a period of time when mastoidectomy is a most injudicious, not to stay a fatal undertaking.

DIAGRAM II.




———Normal total and differential white cells.
- - - - - - Total leukocytes.
..... Neutrophilic granular cells.
******** Eosinophilic granular cells.

If these statements appear ultra-conservative to you, let me qualify them by my firmest conviction of all, to the effect that it is imperative to perform mastoidectomy at the *right time* in *almost all* the cases of panmastoiditis and in *many* cases of partial involvement of the mastoid cells in acute inflammatory disease.

From the point of view of anatomical pathology this opportune interval corresponds to the period of necrosis involving the air cells of the mastoid bone but before the onset of necrosis of the denser bone guarding the lateral sinus, the meninges and the cortical portion of the mastoid.

Without belittling astute clinical acumen leading to the correct selection of this optimum time and appropriate case, it is a matter of such delicate judgment that most of us welcome any aid that presents itself. I am convinced that repeated total and differential counts taken during the development of acute mastoiditis by extension from an acute otitis media, furnishes such an aid.

In speaking of otitis and mastoiditis, Simon has, I think, voiced the general consensus of opinion regarding the leukocytes when he says: "The leukocytes are increased in almost all cases. In the serious form of otitis the hyperleukocytisis is usually slight (10,000 to 17,000) and occasionally absent, while in the purulent cases a brisk increase is the rule (up to 25,000 or 30,000). The same holds good for mastoiditis and complicating intranasal suppuration. In all such cases the differential count will reveal the septic factor. The increase in the neutrophiles may vary from 75 per cent to 98 per cent and is, generally speaking, proportionate to the intensity of the infection and irrespective of the total number (of leukocytes). The same considerations in fact which apply to septic processes in general, also apply here."

I am convinced from experience, repeated in many cases (though not all) that the above statement is inadequate, if not misleading. My own experience leads me to the conclusion that we do not usually proceed from pharyngitis to otitis media, from otitis to mastoiditis and from this to an intracranial complication with quantitative increase in the intensity of the septic factor only, but that there is likewise a significant qualitative alteration in the differential count during the stage of mastoid cell necrosis, separating that important phase from the one preceding it and the disastrous one which follows.

It seems likely that this fortunate alteration is dependent upon the peculiar structure of the spongy portion of the mastoid bone, yielding as it does an extensive surface in a relatively small volumetric compass. The product of necrosis (once again an alien protein) absorbed from this very considerable area, is sufficient to evoke an abnormal number of eosinophiles. A hitherto *"septic factor" is* thus converted into a *"lytic factor."*

Though this sequence of events is not universal, it has occurred so often that it cannot be fortuitous. Indeed, I have come to rely upon the change from a septic to a lytic factor as a valuable guide in determining when and when not to advocate mastoidectomy. In our experience even the exceptions to this rule have worked out fortunately. These exceptions may be grouped as:

*1.* Low-grade infections of the mastoid where but limited area is involved and that effectively walled off. These have a tendency to spontaneously recover. They do not produce a hematogenetic response which includes a lytic factor.

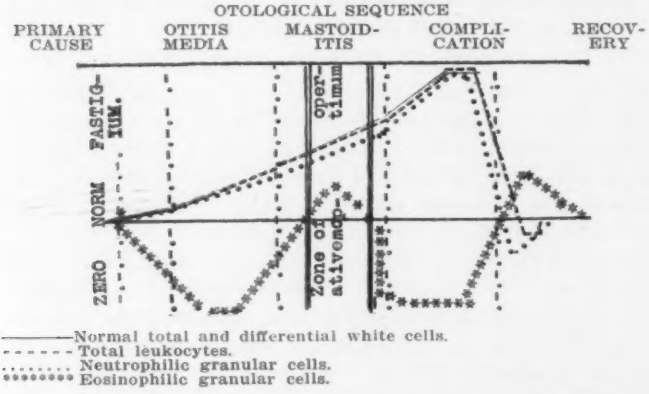
*2.* The initial stage of the average case of mastoiditis. Here the lag in response between invasion and necrosis (it is not until necrosis occurs that hypereosinophilia results) gives opportunity for a pro-

tective inflammatory barrier to wall off the spongy portion of the mastoid from the denser surrounding bone. We feel that this circumstance inhibits too great zeal for the premature opening of the mastoid, a practice which sacrifices a natural ally in limiting the spread of the infection; indeed, not infrequently converts a localized infection into a general one.

*3.* Those especially virulent infections which run so rapid a course that the invading hosts have overcome every obstacle in the path toward a generalized septicemia before any effectual mobilization could be effected. In the face of this type of disease a mastoidectomy is futile. Nothing short of decapitation would suffice.

These three classes of cases which may occur as complications of otitis media do not then alter the conclusion that the optimum time in the development of mastoiditis for operative interference corresponds to the development of a distinctive, hematogenic response—a lytic factor.

DIAGRAM III.




In the event of the further extension of the pyogenic infection to involve the meninges, the lateral sinus or the general circulation, there is so considerable an increase in the neutrophilic granulocytes that the hypereosinophilia is quite overshadowed. There is in this event a reconversion of the lytic factor to a septic factor. Expressed a little differently, the change from a lytic to a septic factor of more intensity than usual should give rise to grave misgivings of the next and often fatal development of either a meningitis, a lateral sinus thrombosis or a septicemia.

In our experience then, the optimum time for mastoidectomy corresponds to an ephemeral blood picture which we designate as a lytic factor, and this lytic factor is interpolated between a mild preceding and an intense following septic factor.

It is my earnest hope that you will tentatively try the plan of making repeated differential and total leukocyte counts in this group of cases if there is any doubt in your mind as to when brement of the mastoid bone should be practiced.

Baltimore Eye, Ear and Throat Hospital.

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## AN IMPROVED METHOD OF SHRINKING THE EUSTACHIAN ORIFICE.*

Dr. Joel J. Pressman, Philadelphia.

Swelling of the torus tubarius frequently accompanies acute infection of the nose and throat, and by closing the orifice of the Eustachian tube gives rise to a sense of fullness in the ear, diminished acuity of hearing and often moderate pain and tinnitus. These symptoms are extremely annoying. Rapid and almost complete relief follows shrinkage of the orifice of the tube.

The usual method of shrinkage is to apply medicaments to the torus by means of a cotton-tufted curved probe carried backward along the floor of the nose to the posterior wall of the pharynx, rotating the curved tip outward and drawing it forward across the fossa of Rosenmüller and the prominence, into the orifice of the tube itself. In the healthy nose this procedure, in skillful hands, is painless and presents no particular difficulty. However, in the acutely inflamed nose, especially in a severe acute rhinitis or hay fever, these manipulations cause much discomfort, and oftentimes pain.

We have recently tried a new method of approach through the mouth that has proven most satisfactory from the viewpoint of results obtained, as well as facility of application. The tip of the

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*From the Department of Otolaryngology, Graduate School of Medicine, University of Pennsylvania.

ordinary DeVilbiss spray is turned upward, slightly beyond a right angle. With tongue firmly depressed with a metal depressor, the shaft of the spray is introduced into the mouth and carried back alongside the uvula to a point just behind the soft palate. It is then rotated so that the tip turns outward through an arc of 45° to 60° into a position in which the spray tip points at the orifice of the Eustachian tube. Air pressure is then gently applied and the shrinking solution sprayed across the soft tissues surrounding the orifice of the tube. It is important to avoid touching the tissues with the spray tip, to prevent gagging. The spray is then withdrawn and introduced on the opposite side of the uvula and the procedure repeated to shrink the second orifice. In the hands of one accustomed to posterior rhinoscopy no difficulty is encountered, and the maneuver can be carried out with no greater discomfort than accompanies an ordinary throat examination.

The advantages of this method of approach are its freedom from trauma to the acutely inflamed and tender tissues of the nose and epipharynx, its freedom from manipulative discomfort or pain, the widespread application of the shrinking solution to the tissues surrounding the orifice, and the avoidance of carrying the probe through an infected area.

There is very little danger of forcing infected material into the middle ear, because the direction of force of the spray is across the orifice and not directly into it. We have not observed any ear complications as a result of this procedure.

Shrinking of the torus takes place quickly and thoroughly, and relief of the tubal symptoms is rapid and often complete.

At times, in noses presenting deformities, making it difficult to shrink the posterior tips of the turbinates, we have found it advantageous to follow a similar procedure and by pointing the tip so that it looks upward and forward in the direction of the choanae to spray the tissues of the posterior nares and produce satisfactory shrinkage. In throats that are not too sensitive, raising the soft palate with the shaft of the spray insures a thorough application.

Occasionally a patient is encountered in whom a pronounced gag reflex prevents the carrying out of this technique, but with increasing experience these cases will be found to be very few, especially if we are careful not to touch the uvula or soft palate with the shaft of the spray.

4043 Baltimore Avenue.

# ALLERGY IN RHINOLOGY.*

DR. ROBERT A. COOKE, New York.

The title of my paper is a broad and comprehensive one and I am sure your secretary intended that I should use my discretion and merely indicate certain phases that are of common interest to the rhinologist and the allergist while dwelling more on others, especially those included in tonight's symposium.

Allergic manifestations of the upper respiratory tract constitute but a small part of the broad field of the rhinologist, just as they also are but a part of the clinical evidences of human hypersensitiveness.

In a general way we can indicate the field of common interest by the assertion that there are:

*1.* Allergic reactions which manifest themselves in part or in whole by nasal pathology and nasal symptoms. These are the most important, but there are also:

*2.* Allergic reactions with general, not nasal, symptoms but due to nasal pathology in the form of sinus infection. These are the cases of bacterial allergy about which we will speak more later. Let me cite here, however, an illustrative case:

Female, age 32 years, had chronic urticaria for eight months. Routine examination revealed a purulent postnasal discharge. The intradermal tests were all negative. Referred to the nose and throat department, a diagnosis of chronic suppurative infection of the sphenoidal sinus was made. Operation was advised and for three days after operation there was a severe exacerbation of urticaria, which then gradually disappeared and, with the establishment of drainage, did not return.

Cases of this type are not too common but they must be kept in mind.

Other general manifestations that may be properly classed as allergic and that may be due to focal infections of the upper respiratory tract are angioneurotic edema, toxic dermatitis or eczema and asthma. In many of these cases nasal symptoms may be elicited on careful questioning, but these symptoms are so minor compared to those for which the patient seeks relief that they are not mentioned.

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*Read before the Section of Laryngology and Rhinology, New York Academy of Medicine, Dec. 20, 1929.

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We have used the word allergy very freely. It has come to stay in medical nomenclature. It is a good word to denote just what it etimologically means—altered reaction. It may be considered synonymous with the cumbersome word, "hypersensitiveness."

Allergy or hypersensitiveness is a state of spontaneous or induced susceptibility. It occurs in a special group of any species which reacts to certain absorbed substances with symptoms that cannot be elicited in the normal of that species.

This allergic state may be (artificially) induced as by the injection of horse serum when it is usually temporary. On the other hand, it may be, as far as we can tell today, a natural or spontaneously developed state, as in hay fever. There are several pathological forms of the allergic reaction, such as simple hyperemia or hyperemia with edema, which is the usual one, or the lesion may take the form of an eczema or dermatitis. The reaction with hyperemia and edema, involving both skin and respiratory mucous membranes, which occurs immediately, that is, within an hour after contact to the excitant, is the commonest and the one about which we know most. These cases can usually be diagnosed by the immediate skin reaction. Antibodies may be demonstrated in the blood of such cases by the method of passive transfer.

Of particular interest tonight are those clinical conditions of the upper respiratory tract which may have an allergic basis, namely:

1. Vasomotor rhinitis (allergic rhinitis) (allergic coryza).
2. Angioneurotic edema involving the upper respiratory tract.
3. Infections of the upper respiratory tract which are secondary to the allergic conditions.

Angioneurotic edema is relatively uncommon and need not be discussed.

The secondary infections of the nasal tract are important. They are by no means uncommon during and after the vasomotor disturbances to be discussed and are also commonly associated with cases of eczema and dermatitis in which the mucous membrane, as well as skin, is involved. The importance lies in the fact that the infectious process in these cases does not get well and stay well unless the underlying allergic condition is recognized and treated.

Of most importance on account of greater frequency are the cases diagnosed as vasomotor rhinitis.

This term expresses a clinical condition characterized by edema of the nasal membrane. Suppuration is not usually present but, if present, is not a prominent feature. What are the causes or exciting

factors of importance in this condition, how is it diagnosed, and what are the principles of treatment?

It cannot be proven that all these vasomotor cases are allergic but the percentage that are allergic is so high that it is conceivable that an allergic phenomenon is the basis for them all.

Included in this group and forming a large percentage of the total are the seasonal pollen sensitive cases with asthma as an associated allergic reaction in 30 per cent. Practically these reactions occur only to the air-borne pollens but some air-borne pollens which are prolific, such as those of cattail and pine, have not as yet been found to give reactions.

Sensitization also exists to the heavier insect-borne pollens of many flowers, such as daisy, goldenrod, aster and sunflower, but symptoms occur only after very intimate contact. They are, therefore, relatively unimportant.

Non-seasonal or perennial cases of vasomotor rhinitis occasion greater difficulty to the rhinologist as a diagnosis or a presumptive diagnosis is not so readily made. Here the patient describes paroxysmal attacks or symptoms may be continuous or continuous with exacerbation. Let us consider these causes more in detail. Our daily and our casual contacts with air-borne substances absorbed by inhalation are manifold and it can be presumed that reactions may occur from any soluble organic and some inorganic substances present in the air. There is the large and well known group of animal emanations—the dander (not strictly the insoluble hair and feathers of all flesh and fowl) and such secretions as the saliva, especially of dogs, and the silk of the silkworm. Air-borne substances of vegetable origin are perhaps more common and comprise the flour of cereals, orris root, cotton seed and flaxseed. This latter substance has recently become quite important on account of its frequent use by hair dressers.

Substances producing nasal symptoms after ingestion are relatively unimportant—certainly not more than 2-3 per cent of the entire group. In infants and young children they do occur, but only as a minor part of an acute general reaction with urticaria and asthma. In older children and adults cases are occasionally found but here too nasal symptoms are usually but a part of a more general reaction. They may occur either to foods or to such drugs as aspirin, arsenic, iodide, quinin, senna, delphinium and others.

The next and last group of causes are the infections. It is not meant to include here all infections of the nasal tract but those in which there is an edematous reaction to bacteria or bacterial pro-

ducts. Suppuration may not be present. The edema of the nasal mucosa is typical of vasomotor rhinitis. Not infrequently in allergic cases, such as hay fever, a bacterial invasion of the upper respiratory tract may evoke a mucus membrane reaction typical of allergy.

*Diagnosis:* Let me now briefly take up a few points in diagnosis of these cases of vasomotor rhinitis.

*1.* There are points in the history that give a clue. *a.* In the type of allergy we are discussing, a positive antecedent family history is obtained in at least 50 per cent. A positive family history is presumptive evidence of allergy in the patient. More commonly, a history positive for asthma or rhinitis is elicited, but it may be a drug or food idiosyncrasy in the forebear. *b.* In the patient a history of food or drug idiosyncrasy or such allergic conditions as asthma, seasonal rhinitis, urticaria, eczema and migraine, past or present, is of importance.

*2.* Our chief reliance in diagnosis is on the cutaneous test. The technique of this need not be discussed. These tests must be used not only with care but they must also be interpreted with something more than a grain of common sense. They should explain the clinical condition and dovetail with the history of the case. For example, a continuous or perennial vasomotor rhinitis is obviously not explained by a positive pollen reaction. A paroxysmal rhinitis is not explained by a positive reaction to a substance to which the patient is daily or continuously exposed, and a reaction *e. g.,* to horse dander in a patient never exposed to horses does not explain the condition. It must be recalled also that there are positive tests which have no clinical significance and there are a few cases of mucous membrane sensitivity in which the skin has been found insensitive and diagnosis may be made by the ophthalmic test.

In some instances we have to resort to the clinical test of environmental change. Do symptoms subside or disappear on change or residence or absence from their usual occupation? One of our cases recently at the clinic was found to be free of symptoms if he stayed away from work—it is not uncommon with furriers—this man was a jeweler and was found to be sensitive to cuttlefish, used in his trade for making moulds.

Another difficulty in diagnosis lies in the fact that something over 50 per cent of these cases have multiple sensitizations—about a third of the pollen sensitive cases react to more than one pollen—and many of the perennial cases are also pollen sensitive. There is apparently no rhyme nor reason in the combinations that are found. Each

individual seems to be a law unto himself and the susceptibilities can be determined only by test, and sometimes not then.

I wish it were possible to give you something more definite and tangible with regard to the diagnosis of the bacterial allergies. In our hands the use of bacterial vaccines for skin tests has not been satisfactory. That this form of sensitization exists is shown by the fact that the use of a vaccine may occasionally reproduce the clinical symptoms, but this happens more often when the skin test is negative than when it is positive to the vaccine. The diagnosis is perhaps best arrived at by exclusion.

It is to this field that our studies are being chiefly directed today but with what outcome the future only can tell.

*Management:* Success in the management of these cases depends entirely upon a correct and complete diagnosis of the cause or causes. Often it is easy; many times it is hard or impossible. It does require patient and painstaking work.

I can only indicate the principles of treatment which guide us in these cases. First and foremost is the avoidance or elimination of the cause. If this cannot be done, treatment by injection of the specific allergen is required. It is not possible to say that these cases are cured by injection. We now resort to a long course of treatment with injections worked up to a point of clinical immunity and we continue injections at monthly intervals for a year or more.

There is a great deal of work still to be done before we shall understand fully the mechanism of the reaction or the nature of immunity from injection. Especially is this true of the bacterial allergics.

In every case the allergist must recognize the importance of nasal pathology.

Cases in which primary or secondary infection exists must be recognized and referred to the rhinologist for appropriate treatment.

Vasomotor disturbances which have progressed to a stage of hypertrophic change usually require operative intervention. Polypi when present must be removed.

Better results, I am sure, can be obtained by a closer co-operation between the rhinologists and allergists in this field, in which they are mutually interested.

60 East 58th Street.

## ALLERGY TO ARGYROL IN A PATIENT WITH CHRONIC PURULENT OTITIS MEDIA.*

DR. ROBERT C. HOWARD, New York.

The patient, a young lady, age about 30 years, came to my office on Oct. 29, 1929, complaining of discharging ears and deafness since childhood, except that the left ear has been practically dry for the past seven years, until three weeks ago, when she contracted a cold, and since then both ears have been discharging fairly freely.

She also stated that her tonsils had been removed seven years before; that she had been troubled all her life with frequent colds and dripping into her throat; that she had never had any nose operations, although she had been treated approximately twice a week for the past seven years with various applications of silver compounds, but more especially with nasal tampons of argyrol, for some years past until about eight months ago, when she discontinued these treatments for five months, and on resuming them she noticed within a few minutes swelling, burning and itching of the lips, face and other parts of the body; together with palpitation of the heart, marked weakness, sweating and other collapse symptoms. These symptoms developed with increasing severity with each succeeding treatment, three in all. The last treatment, the night before she came to me, was so severe as to be alarming and necessitated prompt removal of the argyrol tampons.

On examination I found a septum which was markedly deviated to both sides, but especially to the right, and with enlarged middle turbinates on both sides, blocking the sinuses and the attic of the nose. Pus was present in the nose and on the pharyngeal wall.

Transillumination showed marked cloudiness of the right maxillary sinus.

Examination of the ears revealed large perforations of both drums with canals containing foul-smelling pus. She was very deaf in the left ear, hearing the spoken voice only 12 inches away; and the hearing on her right side was markedly impaired, as the spoken voice was heard six feet away on this side.

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*This paper was read and the patient was presented before the New York Academy of Medicine, Section on Otology, Nov. 8, 1929.

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I advised a nose operation at this time and told her that she might need a mastoid operation on the left side later on and, in addition, would need treatments.

Feeling that tampons of 10 per cent argyrol solution would be helpful in treatment, I was anxious to check up on the suspected sensitization to argyrol, so I suggested such a treatment to her. She readily consented, and I inserted a pledget of cotton saturated with 10 per cent argyrol solution high up in each side of the nose. Within a minute or so her face became congested, swollen and beefy red in color; her hands and body itched so she could hardly stop scratching at them; she complained of weakness, thirst and palpitation of her heart.

I had her lie down, and by keeping her in this position she was able to retain the pledgets for about 20 minutes, when I removed them, after which she shortly went home in fair condition, although quite pale and weak. The following day she came for operation, at which time I performed a radical submucous resection of the septum; bilateral, partial, middle turbinectomy and an intranasal window operation on the right maxillary sinus.

She stood the operation well, and the next day I removed the packing from the nose. She continued to progress uneventfully till the fifth day after operation, when she came for treatment.

ALLERGIC SHOCK, COLLAPSE, ANGIONEUROTIC EDEMA AND DERMATITIS FOLLOWING SECOND NASAL ARGYROL PACK.

The nasal chambers were cleansed with dry cotton wound about an applicator, after which a long, flat pledget of cotton, saturated with 10 per cent argyrol solution, was placed high up in each side of the nose, and very promptly the allergic shock, collapse, angioneurotic edema symptom complex recurred as with the previous treatment, but with increasing severity, and the symptoms became so alarming that I very shortly removed them and had her remain in the recumbent position for some time, until she had recovered sufficiently to go to her home. The itching of her body persisted and kept her from sleeping, in spite of two allonal tablets taken at bedtime. On the following day the dermatitis venenata et medicamentosa (argyrol) on her face was worse; her tongue was coated and she was very miserable. The next day her condition was worse; she had been unable to sleep; the rash had spread from about her nostrils to the lips, cheeks, chin and over the face generally, except for the upper eyelids and forehead, which were not affected. The lymph glands beneath the mandible were enlarged; tongue was more coated; breath more fetid; and the pruritus of the face was so

intense as to necessitate the prescribing of a soothing ointment to relieve the condition.

I decided to do a superficial skin scarification test with a needle, applying 10 per cent argyrol solution to the abraided area and 0.9 per cent sterile saline solution to a similar control area. *Reaction:* Within two or three minutes a very definite erythema was present, and in a few minutes more an elevated blanched wheal, about 1.5 c.m in diameter, became very marked at the site of the vaccination. The reaction rapidly increased up to 15 minutes; remained stationary for 15 minutes more, and then rapidly faded during the next hour.

The skin sensitization test was repeated twice subsequently with substantially identical results.

Superficial skin sensitization test showing negative control area two inches above the large elevated wheal surrounded by wider, irregular, erythematous patch and streaks of lymphangitis about the central argyrol spot.

Under the influence of constitutional and local measures the dermatitis and other allergic phenomena subsided and had about completely cleared up in a week.

Careful search of the literature failed to show any reference to similar reports of anaphylaxis or allergy to argyrol.

Böttner[1], observing anaphylaxis while working with collargol in animal experimentation, made an attempt to connect it with human anaphylaxis.

Stewart and Parker[2], while studying the so-called "endothelial blockade" with collargol, in experiments with guinea pigs and rabbits, noted typical anaphylactic symptoms, which they are unable to explain.

## CONCLUSION.

Although I have occasionally noted, in many thousands of treatments, mild grades of local sensitization of the mucosa of the nose to argyrol, in the form of excessive sneezing and puffiness of the mucous membranes, especially of the turbinate bodies, I have never before seen any reaction with argyrol of an allergic character reaching anything like anaphylactic proportions, nor have I been able to find any such in the literature.

## BIBLIOGRAPHY.

1. Bottner, A.: Ueber Kollargolanaphylaxie und ihre Bedeutung fur die menschlichen anaphylaxie. Deutsches Arch. f. klin. Med., 125:1-22, 1918.

2. Stewart, F, W., and Parker, F., Jr.: So-called "Endothelial Blockade" with Collargol. *Amer. Jour. Path.*, 2:381-99, 1926.

839 West End Avenue.

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## BOOK REVIEW.

**La Rhinite Atrophique et Son Traitement.** By H. Monscourt. 228 pages. Paris: Editions Medicales N. Maloine, 27 Rue de l'Ecole-de-Medecine. Price 20 fr. 1929.

This little volume contains a complete outline of atrophic rhinitis and ozena. The author is rather dogmatic in his statements and assumes as facts many far-fetched theories.

Atrophic rhinitis is considered a local manifestation of a constitutional disturbance and can occur only when nasal physiology is previously disturbed. The author points out a definite relationship between atrophic rhinitis and appendicitis. There is also some connection with the thyroid and genital organs of the female.

The disease occurs only in mountainous regions and always shows a familial predisposition which coincides quite closely with goitre belts.

The treatment of atrophic rhinitis is by iodine and the forms used are carefully explained in all details. The author makes the rather boastful statement that 95 per cent of his cases are curable by his treatment. M. F.

## International Digest of Current Otolaryngology.



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A request has been received from Dr. E. Watson-Williams, of Bristol, England, with reference to cases of papillary granuloma of the larynx, with special reference to occurrence after exposure to mustard or other irritant gas. Information is desired upon the following points:

*1.* Signs, symptoms and clinical appearances. *2.* Date of exposure to gas. *3.* Nature of gas. *4.* Latent period, if any. *5.* Operative measures; except site of granuloma; pathological report. *6.* Exclusion of syphilis and tuberculosis. *7.* Subsequent history.

It shall be greatly appreciated if any of our readers can supply Dr. Williams with notes on this rare condition, together with illustrations and, if possible, microscopic slides. Address all communications to Dr. E. Watson-Williams, c/o Royal Infirmary, Bristol, England.

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Lorelli, in Valsalva F-7-1929, reports some morphological changes in the nose, which were causing mechanical modifications of breathing, blood supply and certain cochlear manifestations. Surgical treatment of the nose relieved all symptoms. Lasagna.

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Humayer, of Budapest, reports that the practice of sewing tampons in the fossa following tonsillectomy results in small hemorrhages through torn blood vessels when the tampon is removed. He suggests the following procedure: He lays a few layers of iodoform gauze flatly over the wound; over this he places an iodoform tampon, and over this the pillars are sewn as usual. Sutures and tampons are removed after 24 hours, leaving only the iodoform gauze on the wound, which usually delivers itself or may be gently lifted off in the course of the next day. This procedure does not change the course of convalescence or healing and rather satisfying results have been obtained in about 100 cases. Kelemen.

Hubert, Arnould and Busser, in the March, 1929, Annales des Maladies de L'Oreille, etc., report a search for tuberculous foci in the adenoid tissue of 200 normal children operated on for usual cases. In every case a tuberculin skin reaction was done two weeks prior to operation. Tonsils and adenoids were histologically examined, and from every specimen inoculations were made into guinea pigs. The writers describe the technique employed and formulated the following statement: Their research failed to discover tuberculous bacilli either in tonsils, adenoids or guinea pigs. From this they infer that primary tuberculous foci in adenoid vegetations and tonsils must be a very rare findings, if every cause of error is carefully eliminated.

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Kelemen, of Budapest, arrives at the following conclusion from his histological studies of congenital syphilis in the temporal bone. A rather frequent picture is one of severe purulent necrotic osteomyelitis of the bony cochlear capsule. The interior of the labyrinth capsule is normal; hemorrhages were found centered throughout, but these are probably terminal manifestations. It is known that such cases of osteomyelitis with bony changes, even when the labyrinth spaces are filled, may heal. This fact accounts for the bony replacement in the labyrinth, which naturally destroys the other parts with their nervous apparatus. Delayed healing is a late manifestation and probably first starts in puberty, which may explain the beginning deafness due to lues, which often manifests itself at puberty. The bony replacement goes hand in hand with the destruction of function. KELEMEN.

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Maurice Sourdille, of Nantes, France, according to a letter appearing in the Journal A. M. A., Feb. 1, 1930, from the Paris correspondent, presented a new surgical technique for the treatment of progressive chronic deafness and for suppurations of the middle ear. For progressive chronic deafness the operation is performed in two stages: the first stage consists in adjusting the mobility of the chain of ossicles; the second stage consists of a depressive trepanation of the labyrinth with immediate closure by a thin epidermic membrane. The operation is said to great increase the hearing ability.

For chronic suppurations of the middle ear, the first stage, that of separation of the tympanic cavity from the mastoid region results in improvement of audition and closure of the tympanic perforation. The mechanism is similar to that of a thoracoplasty which obliterates dead spaces.

Regarding the age at which inflammatory changes in the mastoid are demonstrable by Roentgenologic examination, Martin, in the American Journal of Roentgenology, December, 1929, Vol. 22, No. 5, presents evidence that pathological alterations can be demonstrated as early as six months. He adds that in bilateral affections the interpretation of the films presents much difficulty.

ROSENBERGER.

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Riskin, in the Munchener Med. Wochensch., Nov. 15, 1929, discusses the relationship between scarlet fever and streptococcus sore throat. He points out that it is now almost generally accepted that a streptococcus is the pathogenic micro-organism of scarlet fever and that scarlet fever is not only usually ushered in by sore throat, but that in many cases it presents the same findings in the throat as those seen in what is usually termed streptococcus sore throat.

The author studied the relation between these two during a scarlet fever epidemic. He kept under observation 222 nurses who came in contact with scarlet fever patients; 87 of them contracted sore throat. The length of incubation period in streptococcus tonsillitis was about the same as in scarlet fever. There was a considerable number of cases among the scarlet fever patients who had not been in contact with persons suffering from sore throat.

Riskin found that follicular tonsillitis occurred only once, while the catarrhal and lacunar forms were most frequent.

The Dick test was performed on 91 nurses and it was observed that a negative reaction did not exclude the possibility of contracting a sore throat.

It was suggested by the author that streptococcus sore throat may be an attenuated form of scarlet fever due to an earlier siege of the disease.

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Motta, in Valsalva F-9-1929, describes various mycaeti which he found in cases of ozena. These mycaeti were present in 75 per cent of nasal cases and 25 per cent of throat cases. The penicillium-crustaceum was present in dry secretions, the ormondrendron cladosporicides was found in purulent secretions and the criptococcus when the secretions were mucous. LASAGNA.

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Announcement is made that the Annual Meeting of the American Bronchoscopic Society will be held at Hotel Chelsea, Atlantic City, May 27, 1930. Dr. Thomas E. Carmody, of Denver, is president, and Dr. Louis H. Clerf, of Philadelphia, is secretary of the society.

Linn, in the Nebraska State Medical Journal, Vol. 15, No. 1, for January, 1930, reports that the current throat and ear infections in that locality have been complicated to an unusual degree by adenitis, which has reached almost epidemic proportions. He points out that this complication was commonly reported in the Eastern states during the preceding fall and winter and now apparently is proceeding westward. Standard treatments are advised. ROSENBERGER.

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In the December issue of the Long Island Medical Journal, Vol. 23, No. 12, Smith reports on 100 consecutive mastoidectomies. Probably the most noticeable feature of the resumé is that of the eight radical mastoidectomies, seven were discharged with dry ears. In his experience he rightfully concludes that mastoidectomy is one of the most satisfactory operations of modern surgery.

ROSENBERGER.

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Fox, of Chicago, in the January, 1930, Archives of Otolaryngology, presents a very interesting report of his experiments with camphor, eucalyptol and menthol on the nasal mucosa. His experiments were done on rabbits, and he gives the following conclusions: *1.* A 1 per cent solution of menthol, used over a period of nine months, produces minor degenerative changes; *2.* a 5 per cent solution of menthol causes destructive changes throughout all of the layers of the nasal mucosa; *3.* 5 per cent eucalyptol and 5 per cent camphor have mild deleterious effect on the nasal mucosa when used daily for nine months; *4.* liquid petrolatum also exerts a deleterious effect on the nasal mucosa of a rabbit when used for nine months.

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Cusenza, in Valsalva 10-1929, reports the results of injecting experimentally B. prodigious in the femoral veins of rabbits and dogs. He found that the microbes were collected in tonsil tissue very much as in endonasal lymphoid tissue and several other ways of elimination. The author concludes that the tonsil is of importance for the elimination of bacteria circulating in the blood. LASAGNA.

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Schlenker, of St. Louis, in the Journal A. M. A., Dec. 14, 1929, brings out a new point in the blocking of the internal laryngeal nerve. He puts his needle through the thyrohyoid space until he reaches the thyrohyoid membrane and gently picks up the membrane with the point of the needle and injects underneath. He uses 15 m.m. of alcohol for the injection. MYERS.

## DR. HAROLD GIFFORD.

Dr. Harold Gifford, of Omaha, a graduate of Cornell University, 1879, and of University of Michigan, School of Medicine, in 1882, died suddenly, Nov. 28, 1929, age 71 years.

After receiving his medical degree, Dr. Gifford went abroad for postgraduate study in ophthalmology and otology. He was one of the founders of Omaha Medical College, serving that institution as professor of ophthalmology and otology, Dean of the Medical Faculty, and upon absorption of this school into the Nebraska University of Medicine, he became head of the department of Ophthalmology and Otology until appointed Emeritus Professor.

In earlier years, Dr. Gifford devoted much of his time to laboratory research and made many important contributions to the literature of his special field. In recognition of his scientific and clinical achievements he was honored with the degree of M.A. by the University of Michigan, and of LL.D. by the University of Nebraska.

Dr. Gifford is survived by his widow, two sons (Dr. Sanford R. and Harold, Jr.) and two married daughters.

## SIR WILLIAM MILLIGAN.

Sir William Milligan died Dec. 19, 1929, aged 65 years. He obtained his preliminary education at Aberdeen and studied his specialty in Göttinger and Vienna. He settled in Manchester and was affiliated with the Manchester Royal Infirmary, later becoming Chairman of its Medical Committee; he was also Chairman of the Board of the hospital. He was active in the various scientific societies and held numerous executive offices in these.

His pioneer research in the field of otolaryngology is attested by the numerous references in the standard textbooks. Quite apart from his scientific work, he interested himself in politics, finance, social activities and philanthropy.

During the last two years of his life he was the victim of many severe ailments. He withstood two major abdominal operations, fought two serious attacks of pneumonia and finally succumbed to pneumonia after another severe abdominal ailment.

Sir William Milligan was a brilliant operator, a noted scientist and a beloved teacher. The English-speaking otolaryngologists mourn the loss of a great leader whose demise will be felt throughout the world's otolaryngologic circles. M. F.

## AMERICAN OTOLOGICAL SOCIETY.

*Sixty-second Annual Meeting, May 22, 23 and 24, 1929.*

Hotel Shelburne, Atlantic City, N. J.

*Abstracted by Dr. Robert Sonnenschein, Chicago.*

*(Continued from Page 164, February, 1930, issue.)*

25. When one ear is normal at dip frequency, masking it causes a greater loss by B. C. in the opposite deafened ear, but masking the dip ear occasions little or no loss by A. C. or B. C. in the normal ear. This is probably due to deafness at dip frequency being so great that the masking noise lacks intensity sufficient to cause much masking. It is evident that any lesion producing a tone deficiency area must lie somewhere near the nerve mechanism or the sound conduction mechanism of the ear. In spite of the theoretical possibility of anti-resonance and changes in tension, weighting and friction, many characteristics of these dips and the bone conduction observations make it quite certain that the cause lies in the nervous mechanism of the ear, and the basilar membrane would appear the most promising point for study.

The essayist discussed the possibilities of angiospasm, inflammation, edema, thrombi, emboli, et cetera. It would appear that in many ears there is an element of toxic neuritic or trauma, which may be diagnosed by the presence of the marked deafened area (dip) and which is otherwise unascertainable. "By not testing air conduction at several high frequencies (between 1,000 and 11,000 d.v.) otologists are missing information of the greatest importance. Bone conduction should also be determined at at least one low frequency and at 1,000, 2,000, 3,000 and 4,000 d.v. because by the common fault of omitting these latter, many errors in diagnosis occur."

DISCUSSION.

DR. STACY R. GUILD: I would like to ask Dr. Fowler one question: What is his evidence that the 7 m. point corresponds to the 4,000 pitch in the human being? I am sure high pitches are heard at lower basal end, and low tones at the apical end, where the membrane is broader. I am trying to arrive at a conclusion as to how sound waves become converted into energy, but I do not know yet.

DR. EDMUND P. FOWLER: This is a theoretical arrangement as to the division of the basilar membrane into frequency spaces, and it is about at that distance that theoretically the 4,000-vibration would occur.

DR. GUILD: There is no observed evidence that the theoretical point is correct.

DR. FOWLER: We have endeavored to produce this in animals, but so many unexpected things can happen to laboratory animals. We often have to begin all over again. We have a new set of animals' ears going through the decalcifying solutions now.

DR. GUILD: In regard to the distribution of the gunshot injury in the guinea pig, it does not fall at that point. In the guinea pig the membrane is 18 m.m. long and the area of deafness does not occur in the same place. I used 60 animals in the tests. One ear was exposed and one covered. When plotting out the injured area it was found occasionally as far down as the vestibule.

DR. FOWLER: This 7-m.m. point is suggested from theoretical reasoning, but any other point would be equally satisfactory. In the human subject we have to wait a long time for suitable cases for autopsy, and when we have obtained enough cases with these gaps and loops, and then have them examined post-mortem, we shall be able to speak with more certainty on this question of the 4,000-vibration point.

DR. GUILD: With the audiometer, one man heard 2,048 but did not get 4,096. He was not checked for the intermediate pitches but he fell off somewhere

between the two points. In this ear degeneration was very distinct. There was a total atrophy of the organ of Corti and of nerve fibres in the spiral laminae at the nine to 10-m.m point. Because the lower half of the basal turn is much longer than the rest, we also did monochord tests. The man heard up to 13,000 on the audiometer. When we examined the section there was atrophy, yet he was hearing 13,000. He had normal nerves at the organ of Corti, but he had a patch of degeneration.

DR. A. G. POHLMAN: The first work done on quanititative bone transmission was that by Dr. Kranz and myself. We reported certain lowered acuity around 2,600 d.v. I had a case of lowered acuity which corresponded to Dr. Fowler's tone gaps. The next person tested was normal on one side, and had a total tone gap for bone-transmitted sound on the other. The question was, why he did not hear this with the better ear. Air transmission was normal, but there was a hole in bone transmission. I have the record of a case that was seen by us in which the bone acuity was taken before and after treatment, and showed a distinct area of lowered acuity for bone transmission of sound. Later the bone-transmitted sound gap disappeared. We thought it was a localized lesion of the basilar membrane. It is perhaps somewhat beside the question, but it seems that some of these lowered areas shift and sometimes they disappear, which shows they are not a permanent lesion.

DR. D. W. DRURY: I wish that I had some power with the medical examiner to insist on autopsy of cases that show these types. It is a most interesting problem. I have seen them develop by audiometer records. I think if these cases could be checked up by autopsy it would make a great advance in our knowledge.

DR. ARTHUR DUEL: I would like to recount a phenomenon which recently came to my attention. It was the case of a very well known pianist, who came for treatment for a furunculosis of his ear. He became interested in the tuning fork tests. He told me he had a perfect pitch perception, by which he could always tell when the violin or piano was in perfect tune. He said that the way he determined this was because he knew the pitch was actually always exactly one-third lower than it seemed. In his mind, he always transposes this third and so arrives at the correct pitch. I verified this with the Bezold-Edelmann series of tuning forks.

DR. C. C. BUNCH: The audiogram I show was made from tests of a young lady in whom the right ear was practically normal. Low tones up to 256 d.v. were normal in the left ear but at 512 d.v. there was considerable loss and this increased for tones of higher pitch, the greatest loss being at 1,448 d.v. She heard 4,096 d.v. but did not hear 6,793 d.v. at their maximum intensities; 10,321 d.v. was heard, but 13,004 d.vfl and 16,384 d.vfl were inaudible. With the monochord by bone conduction, she could hear 10,000 d.v., but could not hear 9,000 d.v. or 11,000 d.v. A case like this brings up several interesting problems concerned with theories of hearing. Do these people actually hear sounds at their proper pitch, or is there false perception? In this case, low tones were heard at their true pitch when the two ears were compared, but the tone of 2,896 d.v. seemed in the left ear to be lower than 1,024 d.v., but higher than 724 d.v. It seems difficult to account for this false hearing on the basis of any theory of hearing.

PROF. BENTLEY: The reference to tinnitus reminds me that Stumpf, the psychologist, had a method for determination of the pitch of the tinnitus. Dr. Fowler's reference to the anatomical conditions in his test interested me and reminded me of research done on guinea pigs with long-continued exposure to carefully controlled rates of stimulation. The result of stimulation seemed to be dependent upon the rate of the continued stimulus, as if the rate were the determining factor. If we could go back and determine the character of the sounds, and if this were known, we should know the reason for sudden and continued tinnitus, whether this was the proper vibration rate for the tympanum. We might discover whether there is a certain region of the scale that has a certain sensitivity to shock because of the relation of the tympanic membrane to this frequency.

DR. M. A. GOLDSTEIN: Another phase of this problem is the relation of the peripheral hearing mechanism to the central hearing mechanism. We ascribe

too much importance to the analytical ability of the cochlea or the organ of Corti. For some years I have insisted on tone islands and their recognition through studies by the audiometer. It took me some years to convince physicists that there were tone gaps. I said there was a limitation of the audiogram. I established an arbitrary line, that is, the line of serviceable hearing, and applied that in testing the availability of the audiometer. My claim is that in certain types of serious deafness you may find a hearing capacity below the ability to which the audiometer has been constructed. When you get a tone perception below this limitation, or arbitrary curve of serviceable hearing, we cannot tell whether the patient hears that sound or feels it. It is very difficult to determine where hearing ends and where touch begins. In the work of re-education, where you have tone gaps in the scale, you get gaps of hearing and there gaps and be reconstructed. We know that nerve tissue is the last tissue of the body to regenerate. We don't get frequencies in the cochlea that are lost, but we can get moderate re-education of sounds that are lost. This answers the question that Dr. Duel raised in regard to this mental sound that this professional musician carried in his brain. We have a mechanically perfect machine and an educated sense in the auditory field and these co-operate in the whole problem of hearing. We should not confine ourselves entirely to the consideration of the peripheral mechanism.

DR. GEORGE SHAMBAUGH: This phenomenon is one of the most interesting sidelights on the physiology of hearing and sound perception. The audiometer, as I use it, has its greatest value in throwing light upon physiology, rather than in making diagnoses. Recent, in Chicago, I saw a patient with this phenomenon—three weeks before, quite suddenly in the middle of the day, she had an attack of tinnitus aurium; the ear felt full and there was a defect in hearing. One ear was normal; the other ear had a tone range from 512 to 1,025 d.v., and then dropped off immediately. My interpretation was that she heard nothing above that point. What had happened to that woman? She had not heard a gunshot; she did not use drugs; she had no toxemia. I think it was a circulatory accident, and that an embolus lodged in the labyrinthine artery. That sort of thing might give us some clue to where the point is that the artery strikes the basal coil. Dr. Fowler spoke of these gaps not being found in otosclerosis, but I think they do occur very often in that condition.

DR. J. HOLINGER: This discussion brings to my mind a patient whose findings I presented at the International Congress in Boston. It was a case of diplacusis. The patient was a musician, who heard a different pitch in each ear. I examined him by means of two forks, one held before each ear, changing the pitch of one until the patient heard consonance. The deviation of both ears changed with the pitch, being greatest in the middle octaves. Of course it is a nerve affection, but whether labyrinthine or central is questionable. Diplacusis disables a musician entirely.

DR. J. GORDON WILSON: I would like to ask a question. Ten years ago I was interested in that drop. I found it had a relation to the tinnitus. I thought the position of the tinnitus was higher. I found it nearer the top than the bottom.

DR. J. A. BABBITT: I feel some hesitation in presenting a practical phase on the clinical side of Dr. Fowler's excellent paper, as the previous discussion has been entirely technical. I should like, however, to report a case corroborating his emphasis on the value of routine tests.

This patient, after a long period of treatment by various specialists, came to me with the single pronounced ailment of tinnitus. He was studied by various departments and was seen by one of our associates, Dr. Coates. He was investigated by internists, neurologists, the Barany people and had many laboratory tests. An extensive intranasal pathology was corrected, all to no avail. An outstanding feature in his case, however, was a curious rise of 20 and drop of 40 sensation units in the audiogram at 2,048 and 4.096 d.v., respectively, and this was sustained through all subsequent studies. It was limited to the right side, the side of the tinnitus, and corresponded in general to our analysis of the pitch of his tinnitus, though his tuning fork reaction was untrustworthy. It seemed worth while to record this in the discussion as all evidence seemed to point toward a limited basilar membrane lesion.

Dr. E. P. FOWLER, closing: Dr. Guild's question is very interesting. The 4,000-vibration point is a very rough approximation. I also suggested that above 1,000 to make it as broad as possible. To carry out the hypothesis of hearing, my understanding is that it is not necessary to have the proximal part of the cochlea intact in order to hear the upper notes. There are two ways in which these notes can be supplied in event of anatomical and pathological failure of the proximal part of the basilar membrane. First, if you take a note and cut off its fundamental, the brain will still give you the fundamental, or the mechanism of the ear reproduces it. Second, if the upper note areas are missing, there may be a redistribution of the frequencies along the cochlear membrane. If the upper note areas are absent the next lower areas may be able to function to partially supply the deficiency. The basilar membrane is sometimes bony, not membranous, near the vestibule, and yet the tones of the short wave lengths below may be heard well. Other tones may be heard because of the overtones. In the case Dr. Bunch cites, the area tones were not heard because the tones just below them were not heard loud enough. Dr. Pohlman used the continuous frequency apparatus. I understand that this is not as reliable as the audiometer (I may be wrong), but this would explain the variability in results. I tried for a long time to get a continuous tone audiometer, but the Western Electric people were unable to make it at that time.

In regard to the change of tone sensation, with the limited work I have done I find it is difficult to get untrained observers to distinguish between small changes of intensity and changes of frequency. They mistake one for the other and this easily leads us astray. In regard to Dr. Babbitt's case, in whom there was a good deal of nasal pathology, let me say that many cases do have a sinus trouble, and tinnitus may sometimes arise from nasal pathology. Very often bad teeth cause tinnitus. You may get embolism with acoustic shocks, but I believe a simpler explanation would be embolus of the proximal branch of the artery. That would cut off the upper frequencies entirely. Someone mentioned the keynote of the basilar membrane. It is a very high note, much above the frequency point under discussion. Dr. Goldstein mentioned limits of hearing—we had to stop our tests of the upper limits or get into legal troubles because of possible damage to hearing. Whether or not one can make use of the audibility below the point of serviceable hearing is a question of degree. Above a certain intensity, although there is much sound, there is little definition. Several cases were sent down to us by the Wright Aural School. These children had residual hearing and the question was whether we could teach them to speak. If a child has even slight residual hearing it should be utilized to the utmost. This is an important point in the teaching of the very deaf.

The first case I showed you had displacusis in one ear, and it was lost about half-way up the slope. I did not examine all of the cases for the exact location of the tinnitus. It takes a long time to dig out this information. I examined 12 patients, and in some it was on the lower slope; in some on the upper slope. Often the patients confuse frequency and intensity. If we could get beats, we could get better information as to what is going on in the organ of hearing. In but one or two instances have we been convinced that the phenomenon of beats was obtained.

**Preliminary Note on the Use of Negative Pressure in the Diagnosis and Treatment of Deafness.** Dr. J. F. Fairbairn and Dr. A. G. Pohlman.

Dr. Pohlman stated that it is well known that prolonged occlusion of the auditory tube results in a progressive loss of acuity of hearing, accompanied by retraction of the drum membrane. Some years ago, Pohlman pointed out the degree of hearing loss and the amount of drum membrane retraction were entirely out of proportion to the negative tympani muscle gradually adjusted itself to the very slight drum membrane displacement (took in the slack, as it were) and that this tonus contracture of the muscle, with or without further adhesion, damped out the drum membrane vibrations. He also mentioned the operative possibility of lengthening the contracted muscle by clipping off the processus cochleariformis. The development of the operative technique led to the idea of creating a negative pressure in the external canal to pull the drum membrane lateralward and thereby put the muscle on the stretch and

loosen the parts through fatigue of the muscle. It must be remembered in dealing with sound that the usual amplitudes heard are ultramicroscopic displacements and that a loosening effect equivalent to, say one-hundred-thousandth of an inch, should be sufficient to make a difference in the intensity required to reach the threshold of hearing.

The patient is first carefully tested for air acuity. The authors are employing a beat frequency oscillator with a standard audibility meter shunted across the receiver. The oscillator pitch is plotted at every fifth mark on the dial, which gives 16 intervals in the range from 120 to 8,300 Hertz. The audibility meter gives 33 graded steps of intensity. Next a glass tube is sealed in each external auditory canal with dental impression compound and the entire auricle filled with plaster of paris. The patient is then subjected to a negative pressure of 30 to 40 c.m of water controlled by a water manometer for 10 minutes with occasional oscillation in the pressure of 10 to 20 c.m. The seals are then removed and the acuity of hearing checked. The seals may be used again for a second and all subsequent treatments by applying a liberal coat of vaseline and reinserting. The second treatment is made with a slightly higher pressure of 60 to 80 c.m. of water and it has been the essayist's experience that a greater negative pressure is not required.

Seventy-nine cases of deafness have been treated in our office in Buffalo, with improvement in 30 and subjectively obvious improvement on 21 individuals.

"It is impossible from the limited number of cases studied thus far to determine in advance which type of case will improve and which type will not improve. Neither is the procedure absolutely foolproof."

In a general way cases that report a paracusis Willisii and who respond positively to the acoustic fan test and those with functionally patent tubes seem to react the best. It is also interesting to note that in several cases the low pitch head noises have been completely eliminated, although so far we have found no improvement in alleviating the high pitch sounds.

It is not unlikely that these cases who show every evidence of excellent end organs by reason of the range and acuity of the bone-transmitted sound, determined with a bone activating receiver, are suffering from some sort of mechanical disturbance in the sound transmission system which the delicate negative pressure is not effective.

DISCUSSION.

DR. H. H. VAIL: I have attacked the same problem from a different point of view, but my work is still in the early experimental stages. We have blocked up the external auditory canal with modeling wax and have subjected the animal to compressed air in a compression tank to get positive pressure in the middle ear, rather than negative pressure, which has been described by Dr. Pohlman. Before trying it on my patients I want to see what happens to the middle ear. One rabbit showed small hemorrhages in the mucosa of the middle ear. This treatment which has been outlined by Dr. Pohlman has opened up a new field, which we should investigate. I know that some cases must have adhesions which we can break up by pressure better than by massage.

DR. GEORGE SHAMBAUGH: Were all these cases of retracted drum membrane?

DR. POHLMAN: You cannot tell which cases will be improved. I have had good response in people in whom I did not expect to get any result, and vice versa. In most cases there was a retracted and atrophic membrane. In many cases there was paracusis.

DR. D. HAROLD WALKER: In these cases the tensor tympani muscle is an important factor. Occlusion of the Eustachian tube is not sufficient to account for the retraction of the drum membrane alone. The narrowing of the middle meatus is important also. Where the middle turbinate is squeezed between the nasal wall and septum, this condition may cause a spasm of the tensor tympani muscle.

DR. J. H. FAIRBAIRN: These cases have been investigated very thoroughly. In 76 there was positive right antrum infection and considerable treatment had to be carried on. In cases where the acoustic insult had been caused by a pneumatic drill, we found nothing particular in the nose and throat.

**Some Observations on Anatomical Instructions for Graduate Students Who Are Preparing for Practice in the Field of Otolaryngology.**
Dr. George Shambaugh.

The essayist believes that there is a rapidly increasing tendency toward specialization, so that the problem of providing facilities for men who want to enter our particular specialty stares us in the face. Undergraduate medical instruction for actual practice of the specialty presents more difficulties. It is not take up by the university as it should be, with the result that most men are not adequately prepared. The fact of doing a few operations on the cadaver is no preparation at all. Since the war, the author has undertaken to see what we can do during the first year, in the way of fundamentals. There are two problems to be met: 1. To train men to make proper examination so as to build up sufficient background of clinical experience in order that they may know how to recognize indications for treatment, especially indications for operative interference. 2. The second is the more difficult problem; that is trying to build up for these men an adequate knowledge of the fundamentals in anatomy. This is essential if the man is to be a successful, safe practitioner in otolaryngology. Giving him all sorts of bone to operate on is not enough, because the surgical points of interest cannot be picked up.

The author's plan of instruction is to take 12 men, three each quarter, and have them pay a fee for the supervision. They are employed as clinical assistants. If they pay in advance we are sure that they will not look for other positions in the middle of the course. Half the day is spent in supervised clinical study of patients. The anatomical training consists in the first quarter-year in doing a careful dissection of the head and neck; in the second they make preparations of sections; in the third and fourth quarters they proceed with embryological and with microscopic preparations. The work is different from such work among undergraduates, with whom it is more or less perfunctory. These men grasp every opportunity to work with anatomical preparations. The author has found that the best way to impress ideas on a student's mind is to let him make a drawing, for in this way the idea becomes graven on his memory.

DISCUSSION.

Dr. Edward B. Dench: Anything that will fix the anatomy of the ear in the minds of medical students is a distinct advance in medical education. I know I had considerable difficulty in fixing the gross anatomy of the ear in the minds of my own students. After carefully explaining to them the importance of understanding the normal anatomy of the parts, I give them some operative work on the cadaver, having them demonstrate to me the mastoid operation. In almost every case the students would disregard what had been said about the anatomical landmark for the mastoid antrum and would expose the lateral sinus, or enter the middle cranial fossa. The method which Dr. Shambaugh has suggested, of having the men make tracings in the study of the temporal bone, is excellent. It is quite difficult to copy a microscopic specimen, but it is easier to do than the gross specimen. I should like to bring out here a point which I emphasized in discussing this paper in New York. Sir William McEwen in his book, "Diseases of the Brain and Spinal Cord," mentions a space known as McEwen's triangle. A much simpler method of locating the mastoid antrum, it seems to me, is by a triangle known as the Dench triangle. If we draw a horizontal line tangent to the superior border of the bony external auditory meatus, the aditus ad antrum lies within the triangle whose two sides are formed by the superior and posterior tangent lines and whose base is formed by the curvilinear line of the bony external auditory meatus, included between these tangents. This will locate the mastoid antrum in every instance. The more the operator invades the posterior line, the more likely he is to enter the lateral sinus; the more he invades th suprior line, the more he is endangering the middle cranial fossa.

Dr. D. Harold Walker: In Boston, the Harvard University does not control the eye and ear infirmary. We no longer give a detailed clinical course, but do give a thorough anatomical and pathological one. Dr. Mosher has limited his work to anatomical studies. He is trying to do as Dr. Shambaugh does, to teach by models. He allows the students to make plaster casts in

order to fix the anatomical details in their minds. Dr. Dench's remarks on the antrum are very interesting and embody good suggestions. There is a great danger to the facial nerve by going too near the Fallopian canal, along the posterior canal wall.

DR. S. MACCUEN SMITH: I know of nothing more important than a proper anatomical foundation for those who are preparing for practice in the field of otolaryngology. Dr. Shambaugh's paper is, therefore, especially interesting and timely. We must all agree with the essayist that it is impossible to learn the surgical anatomy of the ear simply by the study of dry bones, but that this must be followed by work on the cadaver, and this in turn by work under careful observation and instruction on the living subject. I think all of us who are conducting large clinics should make it a rule to require our assistants to pass the Board of Otolaryngology, and I further believe that we should not take on new men until this requirement has been met.

DR. L. W. DEAN: I think we can all agree that one of the greatest things which Dr. Shambaugh has done for otology is in teaching the student that fundamentals should be thoroughly known before men can begin clinical work. In out work we have the students make drawings of the preparations, and if there is a case of peculiar interest we let them make plaster casts also. I think this is a very important phase of the work and should have some supervision.

DR. SHAMBAUGH, closing: I spent some time in the infirmary in Boston and saw the models Dr. Mosher is teaching. They were excellent. We do endeavor to give instruction as well. Dr. Sonnenschien teaches them the tuning fork and other functional hearing tests. An operative course on the cadaver follows the anatomical course. We do not allow men in the operating room until the third quarters. They want to watch operations, and would like to operate the first day they get there. We tell them we are not interested in allowing men to operate who do not appreciate the indications.

**Otologic Complications of Swimming in Summer Camps.** Dr. Frederick T. Hill.

One thousand two hundred thirty-five students from 14 selected camps were individually studied. They were all carefully studied at the beginning of the season. Those giving histories or showing any evidence of ear, nose or throat trouble, such as might predispose to otologic complications (36.5 per cent of the total) were grouped, according to the presenting pathology; and restrictions, based upon their clinical pictures, were imposed upon water activities; 1.29 per cent chronic suppurative otitis media and sinusitis cases were advised against any water immersion; 11.17 per cent were placed under partial restrictions, depending upon the conditions. The remaining 24 per cent were not restricted, but were carefully watched by the camp physicians and instructed as to water behavior. These students were followed during the summer and a careful checkup made at the close of the season. No complications were encountered from the first group. There were two cases of acute otitis media from each of the last two groups; a very much lower incidence than in any previous years, and a total absence of any serious cases. There were also five cases of acute Eustachian salpingitis. Practically all of these cases resulted from swimming while afflicted with head colds, the greatest source of ear trouble at these camps. The bacterial content of the water was studied but not found significant. Camp routine seemed to take care of the extrinsic factors, mentioned by Taylor. All types of swimming were practiced, but it was all under careful supervision, especially as to proper breathing. The elimination or restriction of the hazardous cases, showing pathology, or infection, greatly reduced the incidence of complications.

DISCUSSION.

DR. THOMAS J. HARRIS: I have had the privilege of looking over Dr. Hill's paper, and I want to speak in praise of the painstaking investigation which he has conducted. His study of over 1,300 cases cannot help being fruitful in its results. He speaks of the relation of bathing to aural and sinus complications, and this is very important. I think that in this paper Dr. Hill has drawn attention to some of the important economic aspects of this specialty. Dr. Fenton and Dr. Taylor have been giving us the results of their studies for a number

of years, and they point out the fact these complications can occur in fresh running water, where bacteria presumably are absent. The element of fatigue has been emphasized. I want to agree heartily with this point. I think that one hour is a long time to allow boys and girls to stay in the water. Another point that Fenton stressed in his findings is that we should consider the nasal and sinus complications as well as aural disease that may be present. Where there is a latent sinusitis this may be lighted up by a good deal of bathing. Latent bacteria in the nose become active as a result of the bathing, and a latent sinusitis may become anactive condition. The association with the ear is very evident. I have been impressed with the added risk of abnormal conditions in the nose. To illustrate, a deflected septum is a potential factor in causing sinus disease, and is a menace in bathing. What Dr. Hill said about acute rhinitis being a contraindication for bathing is very true. In regard to what Dr. Hill said about diving, I can agree, only I do not think he made it strong enough. I prohibit diving, except one straight dive on entering the water. Out of Dr. Hill's paper as a whole there is one chief conclusion to be drawn, and that is the great benefit of a careful study of the nose, throat and ear of all cases and the preventive measures that should be and are being taken by camp physicians in the elimination of potential risks. Dr. Hill has had only four cases of otorrhea present during the summer. That is a magnificent showing. If we can get camps to recognize this and see the necessity of preventing economic loss, the excellent results which he has presented may be more generally secured.

Dr. J. Holinger: I heartily coincide with Dr. Hill, and believe that this Society should take a stand in regard to two or three points. Firstly, it seems to me that it is in the line of prevention to deafness to make it known that diving is dangerous to the ears, and even to life. The public, and even many physicians, do not know how quickly a person becomes unconscious when cold water enters the ears. Dr. Hill's propaganda is very timely. Another point concerns the method of swimming. The present instruction tends to keep the head too much submerged. Perhaps one cannot swim as fast, but certainly more safely with the head out of water. The constant effort against the water forces the air out of the canal and water takes its place. A third and important point is that in the presence of a slight cold one should keep out of the water altogether, both as regards swimming and the tub bath. True, one feels clearer after a bath for an hour or two, but later on the cold becomes worse and lasts much longer. Writers have called attention to this fact in a number of papers. Any form of hydrotherapy is contraindicated during the presence of a cold.

Dr. S. MacCuen Smith: Most of us have been called upon from time to time to advise whether children should be allowed to swim. Most of the cases that develop aural disease are due to diving, so I feel that this part of swimming should be limited to those of normal ears, and even then the organ of hearing should be protected in the usual way. Most young children are well satisfied if they are allowed to put on their bathing suits, play in the sand, have the benefit of both air and sun, and just before they leave the beach, go into the water without wetting their heads. Unquestionably, much infection is found in the various swimming pools, notwithstanding that every effort is made to keep them clean and as free as possible from bacterial invasion. I served on a committee of investigation a few years ago, and although the water was changed frequently, and in some pools continuously run, the children had "colds," became ill, and with about the usual frequency had ear involvement. I believe that the length of time the children stayed in the pools (and this applies also to bathing in lakes and at the seashore) had much to do with their illnesses and the complications resulting therefrom, from the fact that they became more or less exhausted and this lowered their resistance and recuperative powers. The various rubber devices to keep water out of the ears have been unsatisfactory to both adults and children. The object is better attained by smearing a small plug of cotton with vaseline, placing this in each ear, and then wearing a close cap. The truth of the matter is that the vast majority of cases of aural involvement occur from the attempt of the bather to clear his nose of water, thereby blowing infection through the tube into the tympanic cavity.

DR. J. GORDON WILSON: I would like to ask Dr. Hill what plug he thinks is most efficacious.

DR. D. HAROLD WALKER: I think this is a most timely subject. This is the season when summer camps in New England are in full swing. It is an institution which has come to stay. The children go to camp to learn to swim, and they do not care for camp life unless they are allowed to swim, so that we must give the question of swimming our careful attention. Personally, I believe every person should learn to swim. I should not be here myself today if not for the fact that my daughters have life-saving medals and were able to put their swimming to the test. Dr. Hill spoke of the bacterial content of the water; he mentioned 1,650 per c.c.; that is very high. I think in a case like that there should be an investigation, to determine the type of bacteria. The Maine lakes have very clean water. I was struck by the mention of prior tonsil operations. It shows how much more carefully this work should be done. Fifty per cent of poor results is much too high. I think in giving advice to our patients we should go into details as to how to prevent water from entering the ear. Often in cases where a child has had a dry ear for a long time, he goes to the beach, and after bathing, comes back with a discharging ear. We should show our patients how to use the rubber ear plugs, or vaselined cotton with a rubber cap over the head. I think the time a child is allowed to swim is important. One hour of swimming is very fatiguing. It is the only sport where the body is suspended in a medium without support, and consequently calls for more muscular effort than we realize. I think that if the time were cut down to a half-hour twice daily the result would be much better. I think the question of diving is very important. I had one patient, a young man, who dived off a float. He burst his eardrum, became very dizzy and when in the water lost his power of orientation. Finally he touched bottom and was guided to the shore. It was hours before he got home. He had suffered a very bad rupture from the periphery to the umbo. I have often wondered if accidents of this kind may be a factor in cases of so-called cramps, which so often occurs in wonderful swimmers. In regard to cases of acute meningitis, I have seen one case of death occurring after diving from a high float. The person caught cold, developed sinusitis, and then meningitis. Diving is a very dangerous sport. Swimming is necessary, but diving it not.

DR. F. T. HILL, closing: I heartily agree with what has been said about diving. I feel that the observation made by Dr. Fenton, that many cases of death in the water, so-called cramps, are due to a labyrinthine stimulation is undoubtedly correct. I was talking to a swimming instructor at one of the camps, and while he did not know anything about the labyrinth, he was much opposed to the form of swimming in which the head is submerged, and to plunging for distance. This work was taken up in selected camps and we had pretty good co-operation, but in one camp not on this list in which there was no regular medical supervision we had ten times as much complication. Diving is a menace, but camp authorities will not stand for too much restriction. The same thing applies to fatigue. These youngsters are usually in the pink of condition, able to hike several miles a day, and they will not tolerate having their swimming time curtailed. The average time in camps is an hour, but they sometimes have two half-hour periods. In one lake, where there was a high bacterial content, there was decayed vegetable matter, but no B. coli was found. A fungus growth appeared for the first time last year. These youngsters are often very hard to handle and want to go into the water, whether they have colds or not. The danger, I think, is in the prodromal stage . The danger Dr. Holinger spoke of may be minimized, I think, if the children are carefully supervised and taught to breathe properly when swimming. The question of fatigue is important. People will go to a swimming pool to swim after a hard day's work. This adds fatigue to the risk. As regards plugs, cotton moistened with vaseline or lanolin is preferrable. Rubber plugs might irritate the canal.

**The Use of the New Magnesium Forks.** Dr. Robert Sonnenschein.

Although many devices have been devised for use in functional testing of hearing, tuning forks still remain in many ways the best, most dependable and

easily accessible instruments. Unnickeled forks tend to rust on exposure to the air or handling with moist fingers. Nickeled forks sooner or later give rise to adventitious sounds when the nickeling peels. A complete series of forks, from C-2 (16 double vibrations) to c-5 (4,096 double vibrations) has been perfected by B. E. Eisenhour, of the Riverbank Laboratory, Geneva, Ill., of an alloy consisting of 95.6 per cent magnesium (which has a specific gravity of 1.7), 0.4 per cent manganese (having a specific gravity of 8), and 4 per cent aluminum, with a specific gravity of about 2.7. The alloy, therefore, has a specific gravity of about 2.2; in other words, about one-third that of steel, which is usually 7.7. This metal has two great advantages; first, that it is rustproof; and secondly, that it is light in weight. In testing the lower pitches, the hands soon tire because of the great weight of the low-pitched steel forks, especially those provided with weights. The price of these forks is a little less than that of the best German forks, such as the Edelman series. They do not seem to be any more brittle when properly handled, as all good forks should be.

These forks can be supplied with the "constant" of decrement or damping, and may thus be used for determining the loss of hearing, figured in sensation units, just as is done with the audiometer.

Magnesium forks, just like the medium and low-pitched steel forks, have decided overtones when struck, but these may be liminated by the use of weights, as is done with steel forks, or by placing about the ends of the prongs thin but fairly wide bands of rubber.

The highest pitched c-5 (4,096 double vibrations) magnesium alloy fork does not vibrate long enough for all purposes, but the other forks of the series do very well, both for testing air and bone conduction. When one simply desires to see whether the patient can hear the tone of c-5, the magnesium fork serves very well, but not if one wishes to test actual duration of hearing for this pitch.

Whether the new magnesium alloy forks will ultimately largely displace steel forks in otologic practice cannot be positively stated at present, for it will require considerable experience on the part of many otologists to settle this question definitely, especially with reference to the highest tone, c-5 (4.096 d.v.). It seems, however, that we have here quite a satisfactory solution of the problem of supplying forks made of a metal which is rustproof, and whose weight is such that long-continued testing does not prove as fatiguing as it does with the heavy steel forks. Hence these new forks have some very decided and desirable advantages to their credit.

DISCUSSION.

DR. GEORGE SHAMBAUGH: One point has come up in this discussion, and that is the question of the duration of the vibration of the fork. I prefer a fork that dies out slowly. I do not see the disadvantage in this prolongation of sound. In applying the Rinne test I get a more accurate division and can distinguish better between bone and air conduction. You can note by the duration whether the sound is heard before the ear or behind it.

DR. MACFARLAN: I appreciate being asked to speak before the Otological Society, chiefly because I am interested in forks and the possibility that we will still hang onto the forks. I am enthusiastic about audiometers, too, but I do not see how we can discard forks. I agree in the main with Dr. Sonnenschein, because long experience with forks has brought about recognition of facts which are irrefutable. The Committee has done good work in getting the Riverbank Laboratory to work on the production of a fork which is light and useful, especially when you have to test all the morning. This fork is more desirable than the foreign forks, and I am glad it is an American product. I do not believe that Dr. Sonnenschein brought out the importance of the matter of time. I am less concerned about the matter of pitch than Dr. Sonnenschein is. It does not matter to me whether my forks are 256 or 240, I know the general region in which I am testing. Dr. Fowler developed the fact that there were no very sharp dips in the auditory perimetery. The standardized fork manufactured by the Standard Instrument Co. (the one in which Dr. Mackenzie was interested), was standardized to a very exact pitch. We have no difficulty with the continuous range audiometers in determining the

pitch of the fork. If my steel forks get rusted, I have to renickel them to get the same pitch, but I do not mind if they get a little higher or lower, as long as the hearing time, 55-40 seconds, is the same, because if not, it would alter my record-keeping. When I hit the forks with a pendulum blow the standard time is the same. That gives you a standard hearing time, 55 seconds for air and 30 seconds for bone, which is the normal hearing. In regard to the overtone, fortunately that is not as strong as the fundamental, but Dr. Sonnenschein is right in being on the lookout for the reporting of that one instead of the fundamental tone of the fork. If you make the patient sing the note he hears you can tell whether it is the overtone or not. As to the overtones caused by rust, they are so minute, so transient, that they are of no importance. Rubber bands will give out overtones, but any damping, whether by weights or bands, will not alter workable time. We should allow an interval between testing air conduction and bone conduction. This can be done with a fork that vibrates 150 seconds. The standard Mackenzie forks vibrate two minutes. This takes too long to make the tests. The Alexander forks are 110 a.c. and 70 d.c., but when making a series of tests, 60 a.c. and 50 d.c. is long enough. This gives a faster working time. Another point about the long-time fork is that you are apt to get tone images. This should be avoided, but in children and in adults who have been deaf a long time you are not apt to get tone images. There is another point about the long-time forks, that they die out at the very end slowly. Short-time forks die out very suddenly. The big low-tone forks run a long time with a short vibration stimulus. In regard to the tactile sense, there is confusion in the mind as to whether the patient is reporting tactile sense or hearing, and for this reason I do not use the low tone, or the high fork, but employ the audiometer for the high pitches. In the middle zone of hearing, the forks between 300 and 3,000 d.v. are the forks which give the most useful results in testing. The measurement is by using a standardized blow. The weight of these forks shown by Dr. Sonnenschein is most desirable. I think Dr. Sonnenschein's work with the Riverbank Laboratory has given us something we can thank him for and we ought to be grateful to him for persuading us to hold on to the forks.

Dr. C. C. Bunch: I am glad to see one thing in the new forks which is a real drawback in the Bezold-Edelman forks. I have had graduate students working with a set of forks which I was compelled to use. With some of the larger forks, in order to secure a loud tone, it was necessary to strike the fork with considerable force. The result was that the set screws on some of the forks were bent and broken. To secure repairs was expensive and necessitated cutting the threads with a lathe equipped with gears arranged for metric units. In these new forks the set screw is part of the weight, so that it is not possible to break the thumb screw when unskilled people are using the forks. Secondly, the low-tone forks, especially the 16, make very faint sounds. I think we could make the prong larger to get an increased area of vibration. Some of the earlier reports on hearing have been deficient. It has been said that there was a clinical significance in the raising of the low-tone limit, but if the low tones were made loud enough they could in many cases be heard. It is a distinct advantage to have a fork light enough that it can be handled with the two fingers and thumb. The Bezold forks are very heavy.

Dr. A. G. Pohlman: I agree with both Dr. Sonnenschein and Dr. MacFarlan. Forks have a useful place in testing. All my quanititative measurements of bone transmission are made with a bone-activating receiver with an oscillometer which fits into the manipulator. It is always difficult to plot the lower frequencies. In regard to overtones, I use the 256 and the 512 d.v. fork. In regard to pressure, both bone and air sounds are affected by plus and minus pressures in the ear canal. I would like to ask Dr. Sonnenschein if the laboratory has perfected a method of striking a standard blow. A trip device was designed to give the fork a standard period of vibration. This was graded to activate the fork two or three intensities by a spreader mechanism, so that the fork would be self-activating. This device was for the Lucae type of fork.

Dr. Sonnenschein, closing: Dr. MacFarlan is, I believe absolutely correct in his assertion. It is quite true that slight amount of rusting will not appar-

ently change the pitch of the fork, and it does not make a great deal of difference in testing whether there are 254 or 256 double vibrations. I have always felt that accuracy and uniformity are essential qualities in experimental investigation. We cannot very well compare results unless conditions under which tests are made are uniform. It does not perhaps make so great a difference whether the forks rust or not so far as testing is concerned, but it makes them look unsightly. As for the vibrations of the loose pieces of nickel, even though they may not seriously interfere, why have them if you can avoid them? Another matter is that the renickeling is rather expensive. Our point was simply this: that the Committee of the American Academy of Opthalmology and Otolaryngology (consisting of Dr. Gill, Dr. Mackenzie, Dr. Dean and myself) has endeavored ever since it was appointed in 1921 to educate otologists, especially in the smaller communities, to make the standard tests in a uniform manner. We tried to get forks at a fairly reasonable price. The method of exciting a fork with a pendulum gives uniform results if the pendulum swings through an arc of 90 degrees, the force of the gravity thus being the same each time. I have used a pleximeter, which fell through an arc of 90 degrees, but I realize that a pendulum in many ways is better, and I have at times used the one that was kindly presented to me by Dr. MacFarlan. So far as the rubber band placed around the prongs is concerned, this does not materially interfere with the duration of the vibration of the fork, but it does have a very desirable effect in cutting out the overtones. So far as tone image is concerned, patients do have a memory of tone and therefore it is much better to approach the individual from a distance in testing with a watch, acoumeter, voice or otherwise, and to note at which point he first begins to hear the sound. When a fork vibrates for a very long time, the patient has difficulty in knowing when the sound stops. With the low tones, 16 to 128 double vibrations, the idea is principally to know what the low limit is. We do not as a rule test the duration of the hearing for these forks. We desire to know the lowest tone at which the patient begins to hear by air conduction. With reference to Dr. Shambaugh's remarks about the Rinne test, it often shortens and makes the test more accurate by simply seeing as he does, whether hearing by air conduction or bone conduction is the louder. Dr. Fowler presented some studies on this question about three years ago at the meeting of the American Laryngological, Rhinological and Otological Society in an effort to determine the ratio of air to bone conduction. When the fork is held firmly against the mastoid we naturally damp it considerably and diminish the duration of its vibration. Therefore, it is often better to strike the fork and press it against the mastoid, and see if the patient hears it, then hold the prongs near the ear, then replace on the mastoid, then before the ear, and so on, until the patient no longer hears by mastoid; and then note how long he still hears it by air conduction. The Rinne test shows the difference between air and bone conduction, and one should remember that simply stating whether the Rinne is positive or negative is not sufficient. For instance, there are two forms of the positive Rinne; that is to say, that air conduction is longer than bone conduction, as in the normal, but in those cases in which the air conduction is longer than bone but both factors are shorter than the normal, we usually have an inner ear lesion. Likewise, when we state that a Rinne is negative, meaning thereby that the bone conduction is longer than the air, we should remember that there are several varieties of this, and therefore it is important to know whether the air and bone conduction are considerably shorter than the normal, even though the bone conduction happens to be longer than the air. In new forks made of the magnesium alloy, we have the great advantage of the absence of rusting, and owing to the extreme lightness in weight, the fingers do not tire after doing a number of tests. Furthermore, at the suggestion of Dr. Bunch, clamps or weights were devised which did not have projecting parts, as in the Edelman forks. Some criticism has been made of the fact that the lowest fork C-2 (16 double vibrations) does not produce very loud tones, but Prof. Bentley told me he was surprised to see what sounds could be obtained from these forks in spite of the noise in the room. In striking these forks, a uniform method should be used.

**Traumatic Ear Conditions in Workers Under Compressed Air.** Dr. Harris H. Vail.

The author refers to the monograph of Leonard Hill and to the clinical observations of Alt, Heller, Mager, von Schrotter, Keays and Boot. He quotes a personal communication from the Bureau of Medicine and Surgery, U. S. Navy, to show that from 1924 to 1929 there were no admissions for rupture tympanic membranes in divers. However, the Bureau of Medicine and Surgery felt that the incident of damage to the eardrums among the diving personnel was much greater than would appear from a study of the statistics.

The subjective symptoms which divers and workers under compressed air experience are described and a report from Lieut. Commander G. H. Mankin, M. C., U. S. Navy, is quoted.

The essayist reports two cases of traumatic rupture of the tympanic membrane occurring in caisson workers where the right ear in each case showed multiple perforations which had resulted from pressure not exceeding 30 pounds.

The author's experiments were done on three rabbits and three dogs. The rabbits were subjected to rapid increase of pressure up to 75 pounds, with stage decompression. One rabbit showed no hemorrhages in the middle ear. One rabbit, with one external auditory canal closed by a piece of modeling wax, showed slight hemorrhages in the substance of the middle ear mucosa on that side. One Eustachian tube of each dog was closed by a piece of bougie and the dogs subjected to a rapid increase in pressure, varying from 40 to 80 pounds, followed by a stage decompression.

Gross examination in one dog showed a perforation of the tympanic membrane on the side where the tube was obstructed, with hemorrhages filling both middle ears. The other dogs showed hemorrhages in the middle ears, without perforations of the tympanic membranes. Apparently the presence of the bougie made no difference in the intensity of the hemorrhagic reaction. Microscopic examination by Prof. N. C. Fort showed the mucosa of the middle ears in the three dogs to be lifted off its bony bed and carried well into the center of the cavity of the hemorrhage. In two dogs there was hemorrhage in the vestibule of the internal ear.

The author's summary is that trauma upon the middle and internal ears in workers under compressed air can take place: 1. During compression; and 2. during or after decompression. In the first group the aural trauma is caused by the nonequalization of pressure within and without the middle ear. In the second group the aural lesions are due to nitrogen bubbles forming emboli or areas of necrosis in the internal ear.

The essayist concludes that: 1. A careful examination of the nasal sinuses and ears in workers under compressed air is advisable. 2. Workmen with traumatic rupture of the tympanic membrane should be recompressed only in the event that the symptoms of caisson illness appear, and this should be done under the supervision of an otologist. 3. A wide survey is necessary to decide whether the percentage of deafness is uniformly high in workers under compressed air.

DISCUSSION.

DR. J. G. DWYER: Twenty years ago I autopsied several fatal cases of caisson disease from the Steinway tunnels in New York and found the condition to be that of nitrogen gas in the tissues, the theory being that the cases were due to liberation of nitrogen due to sudden release of pressure, the same principle as release of pressure in a syphon soda bottle.

The present case (described by Dr. Harris) was intensely interesting and along the same lines as Dr. Vail's. The point to be emphasized in this case was the very intense vertigo produced, in contrast to the very slight nystagmus. This would suggest that vertigo and nystagmus do not go hand in hand, as is so often thought.

DR. THOMAS J. HARRIS: This work opens up questions upon a subject which deserves to be very thoroughly investigated. I hope he will not stop before he is able to give us further reports on the internal ear, which are now lacking. The case referred to by Dr. Dwyer was a most interesting one. I have never seen a similar one at the Post-Graduate or the Manhattan Eye and

Ear Hospitals. Dr. Shambaugh says that such cases can offer interesting data as to what is taking place in the internal ear. The patient was an Irishman, age 30 years, newly-arrived. The first time he came out of the caisson he showed marked symptoms of caisson disease. The history was one of intense "vertigo," profound deafness and characteristic symptoms of the "bends." He was subject to spells of weakness, in which he fell and almost lost consciousness. By the audiometer there was complete loss of hearing in the other ear. All tests were negative. There was decided lowering of hearing in the other ear. The graph showed a marked drop for the upper tones, 8,192 being lost. The man was given treatment with pilocarpin and improvement of hearing in one ear was noted, but no material benefit was shown in the audiograms. The man was given cold caloric at 60° and there was no response from either canal. The labyrinth appeared perfectly dead. The man was put in the turning chair and there was a most profound reaction. We only turned him twice and the vertigo which followed was so intense we did not try it again. The nystagmus was very short in either direction. In view of this, can it be possible that we must revise our ideas in relation to the ceter involved? The conclusion that we arrived at was that the man had suffered a hemorrhage sufficient to blot out both vestibular and cochlear parts of the labyrinth. In both sides there were probably multiple hemorrhages. I saw the patient in the hospital before I left and he had recovered from his attacks of vertigo; but the inteersting point is that from the time he came to the hospital, he showed a lowered mental activity. Dr. Dwyer thinks that the mental sluggishness has increased, and that he has reached the point of stupor. We must consider whether the involvement is extralabyrinthine.

DR. SHAMBAUGH: Do you recall any evidence of injury to the middle ear?

DR. HARRIS: The drums were normal. There was no evidence of injury to the middle ear a few days after the accident.

DR. MACCUEN SMITH: You spoke of the administration of pilocarpin. Was the drug given hypodermically, or by mouth?

DR. HARRIS: We felt that the man was entitled to any possible form of treatment. The medicine was given by mouth and consisted of a 2 per cent solution of muriate of pilocarpin, increasing the dose on minim, night and morning, until profound sweating took place. Whether that had any effect on the slight improvement of hearing we do not know. The vertigo has materially improved, almost disappeared, but he is out of the running mentally; he has to be brought to the hospital by his niece. The two points of interest are the discrepancy between the rotation and the caloric test; and secondly, the mental sluggishness. In regard to pilocarpin, Politzer used it in certain cases of labyrinthine involvement, administering it hypodermically. I would like to have Dr. Vail tell us what is the medico-legal aspect of these cases, if he has had any experience with this question.

DR. S. MACCUEN SMITH: It was Politzer who first suggested hypodermic injections of pilocarpin for the treatment of certain labyrinthine diseases. In my experience this line of treatment has been beneficial in a fair number of cases, especially if its administration has been followed by iodin given internally in quickly ascending doses until the physiologic effects have been produced, such as a mild coryza.

DR. F. T. HILL: In 1915, during a period of subway construction, we had some cases at the Manhattan Eye and Ear Infirmary. There were definite symptoms of caisson disease. Some cases have also been seen recently from work on the Canadian border. There is a definite middle ear lesion without labyrinthine involvement. In one case there was a mucoid discharge from the nose and hemorrhage from one ear. Sinus infection would be indicated by the discharge from the nose. This case quieted down rapidly after incision of the membrana tympani, but he was sent back into the caisson before the ears were healed. The hearing was very low on the audiometer. I feel that in a case of this kind it should be insisted upon that the man does not go back under compressed air until the ear lesions are completely healed.

DR. GEORGE SHAMBAUGH: This unusually interesting communication by Dr. Vail has shown the outstanding feature in regard to the involvement of the ear in caisson disease: 1. There is failure to get a proper equalization of

pressure on both sides of the drum membrane; 2. there is liberation of gas emboli into the circulation, with the possibility of their lodging in the labyrinthine vessels. In the first case also we may get labyrinthine involvement. This is quite common in connection with rupture and injury of the drum membrane. It would be interesting to find out whether increase of the pressure, or the liberation of the patient from the pressure is what causes the injury. Both processes might result in traumatism. The patient will recover from a simple tear in the drum membrane, but if there is hemorrhage into the internal ear there will be Meniere's syndrome, with permanent deafness and tinnitus. In regard to the air emboli, I know a case where a man was in the habit of loosening up the septal membranes by air pressure. In one case the patient developed Meniere's syndrome and the hearing was wiped out in one ear.

I hope that the studies made in this disease will add to the acquisition of knowledge of the physiology of the internal ear. If the facts are kept clearly in mind we may get some very interesting sidelights on the function of the internal ear. The labyrinthine artery is a single vessel and if that is plugged all function of the labyrinth is wiped out. On the other hand, minute gas emboli need not block up the whole labyrinthine vessel. The first branch supplies the proximal two-thirds of the basal coil and also part of the ampulla. If the lesion is located here the high tones should be lost by audiometer tests. We know how far the artery extends, and a lesion of the basil coil should produce disturbance of tones and also upset the function of the posterior semicircular canal. That might tell us something of the function of the posterior semicircular canal. The second branch supplies a definite area; the macula of the saccule; the macula of the utricle; and the ampulla of the posterior and superior semicircular canals. In questions where we can exclude middle ear involvement, we may be able to differentiate between labyrinthine and cochlear involvement. According to the involvement of the vessels of the modiolus, the apex of the cochlear may remain intact. The vessels of the modiolus are in the nature of end-arteries and do not communicate with neighboring blood vessels except by minute branches under the tunnel of Corti. The vessels running along the lamina spiralis ossea supply Corti's organ, and their occlusion would produce a lesion of the organ of Corti. Study of these cases should give us insight into the physiology of the organ of Corti.

Dr. J. Gordon Wilson: When you did the caloric test, did you put the head back; in other words, was it done on both the superior and the horizontal canals?

Dr. Thomas J. Harris: That question was asked of Dr. Dwyer at the meeting of the Academy of Medicine. The caloric test was done in the various positions.

Dr. Robert Sonnenschein: I would like to ask the question how it is that if there was no reaction to the caloric test with the head in the various positions, there was, nevertheless, found to be some response of the canals on turning, and this response was equal in both directions. If the caloric response was gone, how can one explain this phenomenon?

Dr. Harris: We did not know how to reconcile the discrepancies. The chief thing was the profound vertigo. It looked to us as if we would have to revise our views as to the mechanism of nystagmus. We hope to make a further report on this case later on.

Dr. J. Gordon Wilson: We are not justified in saying that all vertigo is of aural or even vestibular origin. The profound vertigo to which Dr. Harris refers does not astonish us. The patient showed a markedly affected mentality and it is characteristic of cerebral disturbances that they give pronounced vertigo. This can be often seen in neurasthenic patients complaining of dizziness. The explanation appears to me to be that in such cases a cerebral influence is lacking, preventing the normal inhibitory influence from the cerebrum.

Dr. Sonnenschein: Alexander would not coincide in that view of nystagmus.

Dr. Wilson: You can get vertigo without nystagmus. It is well exemplified in the dizziness which some people experience on looking down from a height.

DR. H. H. VAIL, closing: The discussion has done what I hoped it would do; namely, to bring out the important aspects of a problem which is a growing one. Undoubtedly there will be many works of construction, and this condition will recur in the future. I am glad Dr. Shambaugh said what he did. There is no report made of examination of the internal ears, and no microscopic study made of the ears or temporal bones in the cases autopsied. That should make us watch for these cases and go after an autopsy. Until you have a final proof you do not know what happened. Dr. Dwyer's case was an interesting one, but there are many similar cases reported in the literature. A man was made deaf, blind and speechless by caisson disease. I believe the only treatment is prompt recompression as soon as symptoms appear. The laws of New York State specify the length of time a man can work under pressure, and give regulations as to decompressions and recompression. It is foolish to take cases to the hospital unless there is a recompression tank there. The time to beat the case is when the symptoms first appear. Recovery may then take place as the bubbles of nitrogen are recompressed back into a soluble condition. There is a very definite legal aspect to this problem, because most of these are industrial cases. New York State has a definite code and most other states are furnished with New York laws in construction cases, but I think the construction companies are often cold-blooded, and do not insist upon otological examination. They should be forced to provide these for the workmen whenever necessary. Otologists should be readily available because the ear lesions in caisson workers are serious. The ear troubles in the two cases I have reported are due to compressed air, and are probably not due to nitrogen bubbles. In regard to Dr. Shambaugh's remark about the elecation of the septal mucosa by compressed air in performing a submucous resection of the septum, with formation of an embolus, that is entirely different from the embolus in caisson disease. This forms in the spaces where circulation is poor, such as the spine and joints. The brain usually escapes. Dr. Dwyer's case is one of cerebral damage. Sometimes the brain and cerebellum escape but the spinal cord does not. Often these workmen get into this difficulty from inexperience. In France an aviator came down too quickly and bled from the ears. A few days later there developed mastoid tenderness and a mastoid operation was done. The middle ear and mastoid were filled with blood, caused by hemorrhage on passing too rapidly from the lower pressure to the higher one.

## THE PHILADELPHIA LARYNGOLOGICAL SOCIETY.

*Regular Meeting, Tuesday, Nov. 5, 1929.*

**The Lingual Tonsil.** Dr. M. Valentine Miller.

*(To appear in a subsequent issue of* THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. MARGARET BUTLER: Several years ago, I had a patient with an abscess as large as an English walnut under the lingual tonsil. The patient had gone to the hospital, suffering with extreme dyspnea and I was called to see him.

My first impression on observing him was that he had a peritonsillar abscess, but on using the tongue depressor, no swelling was apparent in the oropharynx. The laryngeal mirror, however, revealed tumefaction as large as an English walnut, occupying the left half of the lingual tonsil. On palpation this was found to be fluctuating. It was incised and a large quantity of pus discharged.

DR. COHEN: About 12 years ago I was compelled to look up this matter and found little literature on the subject. I was doing some work at the Tuberculosis Home and found that so many of them had a varix of the lingual tonsil region. A little later on a baker called to see me about a cough. I examined him and found he had a mass enlargement of the lingual tonsil and after removing that with Myles' lingual tonsillectome he lost his cough.

In people who have cardiac conditions or people who are markedly constipated, we often find a varix of the lingual tonsil. Bakers or people who work on engines, stokers are those who are apt to have an hypertrophy of the lingual tonsil, due to the inhalation of the hot air.

I recently healed a patient with Vincent's infection confined to the left half of the lingual tonsil. He had been treated by a general practitioner for sore throat and after a week he came to see me. With a laryngeal mirror I found a beautiful gray-white patch in the region mentioned. By that time the tissue was eaten away. He had a positive case of Vincent's but was negative to T. B. The case cleared up in about 10 days, after which I sent him to a dentist, who found that he had the same condition in his gums.

I had a case of gumma of the left lingual tonsil in a lady, age 65 years, which I first thought was malignant. From the history of the case, I found she had a son suffering from arthritis. After some persuasion, we gained permission to make a blood test on the son and found he had plus four blood. Had a Wassermann made on the lady and hers was also positive. She got well after proper treatment. It shows how important it is to go into the history of each individual case.

DR. COATES: I think it would be a good thing for this Society to appoint someone to read a paper on the lingual tonsil every three or four years and bring it back to our notice, because we always forget about it. It is surprising how often it is overlooked. The general practitioner does not know it exists.

We have acute conditions and chronic infections. I wish to recommend the work of Dr. Thomas R. French, of Brooklyn. Dr. French did most of his work on the lingual tonsil when he was over 70 years of age, and he has brought it more clearly to my mind than anyone else. He has brought out just how much infection can be there. We take the tonsils out and we usually take the adenoids out fairly well, but we neglect the lingual tonsil entirely. It is not customary to entirely remove the lingual tonsil, but it is most important to keep this matter before our minds and the minds of the younger men.

In regard to the men that do not make laryngeal examinations, it is extremely important that we should study the base of the tongue, as well as the larynx. Chronic infection may and often does start at the base of the tongue.

DR. BURNS: I recall, in my younger days, in the old Polyclinic Hospital, a case that died—a result of a condition that first infected the lingual tonsil. At the start we thought it might be Vincent's, then anthrax. My chief, Dr. Watson, Dr. Walter Roberts, Dr. Freeman and Dr. Kolmer, all saw the patient. It was negative to a Wassermann test and it went on eating itself away until it was entirely destroyed. There were no hemorrhages. Even with such distinguished men, we had to eventually decide it was an undiagnosed

case. The patient first complained of a fullness of the throat, and the practitioner who saw him had become alarmed. If you know Dr. Watson, Dr. Kolmer or any of these men and their ability, you can very well appreciate what a condition it was that this man passed out as an undiagnosed case.

Dr. Miller referred to T. B. of the lingual tonsil. I have had 16 years' experience and have never seen a case of active tuberculosis of the lingual tonsil. We have found that the majority of cases of pulmonary tuberculosis, who show an excessive secretion in the nasal pharynx, do have an enlargement of the lingual tonsil. We do not usually operate on these cases. We have a large clinic and we have devoted very much time to the treatment of tuberculosis.

I enjoyed Dr. Miller's paper very much. It is very worth while to encourage such work among our younger men.

DR. SHUSTER: The greatest part of my experience with the lingual tonsil was in cases that complained of coughing. This occurred not only in tuberculous patients but also in others as well. Occasionally you get patients to be examined for the cause of a cough, the internist not being able to find any. Invariably, in cases of hypertrophied lingual tonsil, where there is a cough, the larynx actually touches the tonsil. When there is actual contact the cough is produced. Good results are obtained by snipping the tonsil off with a Myle's lingual tonsillectome. When not very large, cauterization with a 10 to 20 per cent solution of silver nitrate will be effective. Frequently, the galvanocautery is useful.

DR. MILLER, in closing: I do not have much to add. I find that when the lingual tonsil is enlarged and causing a cough through irritation, it gives the patient great relief to allow an anesthesine lozenge to dissolve on the tongue from time to time. This will frequently allow a patient to carry on with comparative comfort until such time as more radical treatment can be instituted. The use of these lozenges after surgical removal, cautery or electrocoagulation will give great relief and will permit the patient to swallow food when it would otherwise be very painful.

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*Regular Meeting, Tuesday, Dec. 3, 1929.*

**Report of a Case of Focal Infection Neurolabyrinthitis Manifesting Nystagmus of a Character Assumed to be of Purely Cerebellar Tumor Origin.** Dr. Geo. W. Mackenzie.

*(Appears in full in this issue.)*

DISCUSSION.

DR. SHUSTER: I think the statement that Dr. Mackenzie emphasized in regard to pathognomonic signs in intracranial lesions is most important. In this particular case, if Dr. Mackenzie had at once felt sure, he would have made a diagnosis of a cerebellar lesion after his first examination, but Dr. Mackenzie studies his cases thoroughly, and further evidence precluded this diagnosis.

From the description of the case, the diagnosis of nystagmus was based on a lesion in one ear. On the other hand, it is conceivable that a focal infection may attack both sides unequally and we get a confused nystagmus. One can produce almost any form of nystagmus by stimulating the proper canals and placing the head in the proper position.

DR. SHEMELEY: I had the pleasure of assisting Dr. Mackenzie in making these findings, by assuming a rather uncomfortable position upon the floor while I observed the nystagmus. I won't retrace the conditions for which we originally examined the patient for nystagmus, but there was no question about its character.

This is a rather difficult paper to discuss. When a case is presented by such a careful observer as Dr. Mackenzie, we accept his findings and there remains but little to discuss.

DR. MACKENZIE, in closing: I wash to thank Dr. Shuster and Dr. Shemeley for their discussion of my paper. I appreciate Dr. Shuster's remarks, since all that he has said is true.

He speaks of focal infection affecting both sides. When bilateral, one side may be affected more than the other, resulting in a confusion of findings, so far as the direction of the nystagmus is concerned. The cases of focal infection neurolabyrinthitis that I have seen have been practically all unilateral cases. On the other hand, Dr. George E. Shambaugh, of Chicago, several years ago, reported three cases of bilateral involvement from tonsillar infection; but up to the present time I have not seen many cases of this kind. I find that neurolabyrinthitis is most often due to focal infection about the teeth, in which case the involvement is practically unilateral.

In the study of vertigo of labyrinthine origin, the technique of the examination is most important. For instance, if an individual has a very mild grade of nystagmus to the right side when looking straight ahead, it is possible that this nystagmus could be overlooked if the observer is careless and permits the patient to look somewhat to the left of the middle line, when the physiolocal pull to the left side overbalances the vestibular pull to the rgiht side.

As for the estimation of the intensity of the nystagmus when looking to the right and to the left sides: If the observer has the patient look 70 degrees to the right side and 50 degrees to the left, the patient will manifest a greater intensity of nystagmus to the right side than to the left and he will conclude that the patient has a hyperirritability of the right labyrinth, or a hypoirritability of the left, all due to faulty technique in making the observation.

The study of nystagmus is a very interesting and fascinating one, which requires very careful technique.

**Xanthoma of the Larynx.** Dr. Harry A. Schatz.

*(To appear in a subsequent issue of* THE LARYNGOSCOPE.*)*

DISCUSSION.

DR. GREENBAUM: I presented this patient before the American Dermatological Association in May, 1925, and the case report was printed in the Archives of Dermatology and Syphilis, January, 1926.

I have thrown upon the screen a photograph of this patient taken four years ago. The yellow xanthomatous nodules are visible here at the left buccal commissure, in the nasolabial furrow and at the inner canthi of both eyes. Here, just above the upper lip and just below the lower lip, is seen a diffuse, yellowish thickening. The present appearance of this boy is considerably altered from what you can see in this photograph. His lips are drawn together, as are both eyelids, and he is considerably emaciated. The eunuchoid voice mentioned by Dr. Schatz was present when I first saw him, and is still present. The hoarseness is new and is the result of the xanthomatous infiltration into the epiglottis and larynx. For a long time the condition xanthoma was a mysterious disease. A number of years ago, following the discovery, in some of these patients, of a hyper-cholesterolemia, the theory was suggested that xanthoma was the result of cholesterol infiltrations into the skin and other tissues. However, a new theory has recently been evolved by Udo Wile, which seems to be nearer the truth. As the matter now stands, the condition may be viewed in the following way: Xanthoma is to a certain general condition termed xanthosis what the gumma is to syphilis. Xanthoma lesions are therefore likewise manifestations of a general process. This general process is in all probability related to the fats in the same way that the sugars are to diabetes and the proteins to gout. The actual infiltrations consist of fats and lipoids, and although they occur in the skin, they likewise occur, and probably with greater frequency, elsewhere in the body. This is notably true in cardiovascular system in the form of atheroma, in the eyes in the form of arcus senilis, and they of course occur in mucous membranes. These metabolic disturbances may be mixed, since xanthoma occurs in diabetes, in hepatic disturbances, with or without the presence of lipemia and with or without a hypercholesterolemia. The prognosis is poor in the type known as xanthoma-tuberosum, of which this case is an exemple, but is decidedly better in the types xanthoma diabeticorum and in the type xanthoma palpebrarum, which occurs in form of yellow plaques around the eyelids, such as you see here in this slide.

## EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION.

*Regular Meeting Held at Troy, N. Y., Dec. 19, 1929.*

**Maxillary Sinusitis Cases Studied by Means of Lipiodol Injections.**
Dr. Marvin F. Jones.

Dr. Marvin F. Jones, of New York, gave the clinical histories of some 50 consecutive cases, not selected, in which the clinical histories justified a diagnosis of antrum of Highmore disease of such a chronic and complicated type that investigation by lipiodol injection studies were indicated.

Each case was illustrated by Roentgenograms before lipiodol injection, immediaately after lipiodol injection, and follow-up Roentgenograms showing end-results after irrigation, antrum drainage by antrotomy and by radical antrum operations, according as each or all of these surgical procedures were indicated. The series of Roentgen studies were made by Dr. Marvin F. Jones and Dr. Perry Lund.

The following technical points were brought out:

In making lipiodol injections, a local anesthetic of 30 per cent alypin applied at the point of selection for puncture.

A fine Ruskin needle is used, and care is taken to insert the needle beyond any intra-antral obstruction or edematous granulation or polypoid tissues.

The antrum is then irrigated with lysol solution; this is followed by attach-a piston syringe (with the plunger graduated in c.c.) to the needle in situ. Any undue force is not permissible, as it generally means that the needle tip is not free in the antrum cavity, but is blocked by intra-antral tissue.

The lipiodol solution used consisted of lipiodol one part to sterile olive oil five parts. The solution is injected until it escapes through the ostium or returns along the needle shaft. The amount varies from 1 to 5 c.c.

The Roentgenograms are made with the patient in the sitting position.

The various filling defects of the lipiodol shadows were clearly shown by anteroposterior and lateral stereoscopic Roentgenograms viewed by means of hand stereoscopes.

After injection of the antra, Jones and Lund make the stereoscopic Roentgenograms immediately in their own offices. However, they state that this is not necessary as the lipiodol injections have a long residual time in the antra and although they consider their series of cases too small to fix the residual time of lipiodol in the antrum, Dr. Jones estimated that the normal residual time was as long as one week.

No poisonous effects have been noted from the lipiodol oil one to five solution.

The techniques of antrotomy and radical antrum operation were described. Drainage in antrotomy is maintained by means of a self-retaining rubber catheter until epithelialization is complete.

DISCUSSION.

DR. J. IVIMEY DOWLING: Dr. Jones deserves the thanks of this Society for bringing to the attention of the members so valuable an aid in establishing diagnosis of antrum disease.

Accuracy in diagnosis leads to appropriate treatment or surgical procedure and so we may well consider the injection of lipiodol into the antra as well worthy a trial in the cases that fail to yield to the usually successful methods.

I have had no personal experience with this procedure, but while at Battle Creek Sanitarium I was decidedly impressed by the work of Fraser.

It has been my privilege to have witnessed the progress of rhinology over a period of many years. In the early days this special surgery was crude and ofttimes left the patient worse than his original state. With the advent of adrenalin chlorid there was a great advance made in all surgery of the sinuses, and from the primary crudities there has evolved a special rhinological surgery

that will bear comparison with the efforts of all surgeons, and anything that will help to aid us in better diagnosis and improve our surgical technique is worthy of investigation, and so I believe we will all benefit because of Dr. Jones' able presentation of this method.

DR. A. W. GREENE: It has indeed been a pleasure to listen to Dr. Jones' splendid paper and see his really splendid series of Roentgenograms. I have used lipiodol on occasion but feel now that I can make more and better use of it.

It might not be amiss for me to cite one very interesting case in which I used lipiodol:

A few weeks before this patient came to me he had the left upper premolar removed. A fistulous tract, discharging a foul pus, remained. I was able to insert a probe an inch-and-a-half, but attempts to irrigate and have the solution come through the nose failed. I then inserted a Lichwitz needle under the inferior turbinate but go no pus. I then attempted to touch the Lichwitz needle with a probe through the tooth socket but failed. An X-ray showed that they should be touching.

Lipiodol injected into the sinus through the Lichwitz, surrounded a balloon-shaped mass, in the center of which the probe could be seen, according to a second X-ray. Diagnosis was made of granulation tissue or abscess wall which had formed above the root of the tooth.

A Caldwell-Luc verified this diagnosis. This hollow mass was curetted away from the tooth fistula; a large counter-opening made in the nose, and final cure.

Without lipiodol diagnosis would have been pure guesswork.

DR. JOHN J. RAINEY: Several years ago a woman, age 55 years, came to me, complaining of nosebleed and swelling over the right side of face. Polypi were found in right side of nose, which bled at the slightest manipulation. Biopsy showed basal cell carcinoma.

A fistula was discovered external to the root socket of the second right molar, the tooth had been removed with great difficulty five years previous. The patient felt that her symptoms began after the extraction.

Antrum washing and X-ray examinations by Dr. Rowan were negative.

An X-ray with the probe gently introduced to its limit, showed the probe was well in the orbit.

Lipiodol was gently introduced through the fistula. X-ray showed the soft tissues of the face involved, the antrum negative. The disease which involved the orbit through the soft tissues had broken through the lamina payracea to the ethmoidal labyrinth, with subsequent basal cell polypoid degeneration.

She had a severe orbital reaction to the lipiodol, but in a few weeks it had entirely disappeared.

Dr. E. A. Stapleton, Dr. F. S. Sulzman, Dr. A. F. Holding and Dr. John J. O'Keefe brought out points of technique and interpretation of lipiodol Roentgenograms.